DISSERTATION ON

A STUDY ON THE PREVALENCE OF INSULIN RESISTANCE IN YOUNG ADULTS WITH ABNORMAL MENSTRUAL PATTERN

Dissertation submitted
in partial fulfillment of the regulations
For the award of the degree of

M.S. DEGREE - BRANCH - VI

OBSTETRICS AND GYNAECOLOGY

Of

THE TAMIL NADU
DR. M.G.R. MEDICAL UNIVERSITY

ESIC MEDICAL COLLEGE &
POSTGRADUATE INSTITUTE OF MEDICAL SCIENCES AND RESEARCH
KK NAGAR

APRIL - 2014

CERTIFICATE

This to certify that this dissertation **"A STUDY ON THE PREVALENCE OF INSULIN RESISTANCE IN YOUNG ADULTS WITH ABNORMAL MENSTRUAL PATTERN"** submitted by **Dr.R.RAJESWARI** appearing for M.S. Degree, Branch - VI **OBSTERICS AND GYNAECOLOGY** examination in April 2014 is a bonafide record of work done by her under my direct guidance and supervision in partial fulfillment of the regulations of the Tamilnadu Dr.M.G.R. Medical University, Chennai, Tamilnadu, India.

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DEAN

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DECLARATION

I solemnly declare that this dissertation entitled **"A STUDY ON THE PREVALENCE OF INSULIN RESISTANCE IN YOUNG ADULTS WITH ABNORMAL MENSTRUAL PATTERN"** was done by me at ESIC Medical College & PGIMSR, K.K Nagar, Chennai during 2012 - 2013 under the guidance and supervision of Professor **Dr.T.K.RENUKA DEVI, MD., DGO.** and Associate Professor **Dr.KANAHESWARI, M.D., DNB.,** This dissertation is submitted to the Tamil Nadu Dr.M.G.R.Medical University towards the partial fulfillment of requirements for the award of M.D. Degree in OBSTERICS AND GYNAECOLOGY (Branch - VI).

Place: Chennai-78.
Date

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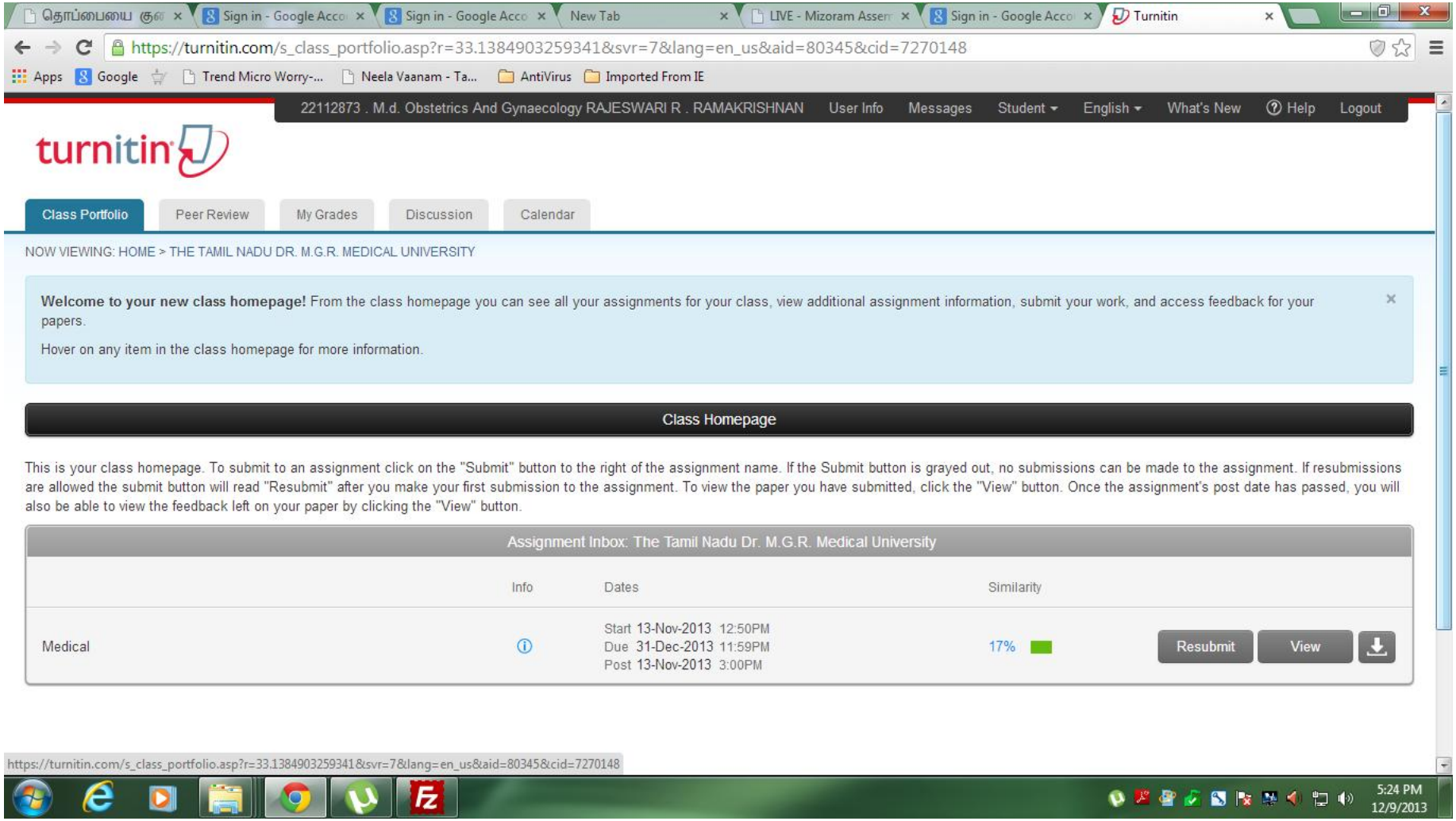
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ABSTRACT

INTRODUCTION:

Anovulatory bledding reflects an abnormal pattern of steroid hormone stimulation that deviates from the sequence characterising the normal ovulatory menstrual cycle.

Polycystic ovarian syndrome is a common, heterogenous disorder of women of reproductive age characterised by chronic anovulation, hyperandrogenism and insulin resistance.

Insulin resistance is associated with an increased risk of various disorders like Type 2 Diabetes mellitus, Hypertension, Dyslipidemia, Heart disease, endometrial cancer, recurrent miscarriages and pregnancy complications like GDM, Preeclampsia.

Timely intervention could perhaps prevent or delay the onset of these conditions.

Key words: Polycystic ovarian syndrome, oligoovulation, insulin resistance

STUDY DESIGN / METHODS:

This cross sectional survey with nested case control study was done in the Department of Obstetrics and Gynaecology, ESI Medical College and PGIMSR ,in association with 11 health camp visits conducted at an export factory, MEPZ, Tambaram, Chennai done

between August 2012 – August 2013.These subjects were ESI beneficiaries.

A total of 223 young adults were recruited after informed consent and a questionnaire on the menstrual pattern of adults working in the export factory. Among them 129 with long cycles / scanty periods were in the test group and 93 in the control group. More number of subjects was enrolled in the study group to increase the accuracy of the study and due to availability of subjects and after the consultation with a Statistician.

All subjects had a detailed clinical examination, questionnaire for stress scale score, biochemical investigations, and ultra sonogram. Among them 68 met the criteria for PCOS fulfilling 2 of the three of Rotterdam criteria. These subjects had oligo ovulation and ultrasound criteria of Bilateral PCOD.

As per the Aim of the study insulin resistance was calculated first for the 129 with long cycles and scanty periods and then for those with Polycycstic ovarian syndrome.

RESULTS:

There was a 7% prevalence of insulin resistance in individuals with long cycles / scanty periods

There was a 13.2 % prevalence of insulin resistance in those who met the criteria of PCOS

There was a 71.8% association of stress with long cycles / scanty periods and 95.8% association of stress in those with PCOS.

CONCLUSION:

It is observed from the present study that insulin resistance manifests at an early age in women with polycystic ovarian syndrome. Oligoovulation (Long cycles / scanty periods) is a useful marker to identify subjects with insulin resistance.

Counselling regarding Life style modifications, maintaining BMI should be made available to all women who are insulin resistant.

Thus by early diagnosis ,at an asymptomatic stage progression of the disease can be halted.

CONTENTS

INTRODUCTION

Anovulatory bleeding reflects an abnormal pattern of steroid hormone stimulation that deviates from the sequence characterising the normal ovulatory menstrual cycle[(1)].

The normal limits for duration, regularity and frequency of menstrual flow were based on the 5th and 95th percentiles for data drawn from population studies and as such common anovulatory disorders such as Polycystic Ovarian Syndrome influence these data.

Polycystic Ovarian Syndrome is a common, heterogenous disorder of women of reproductive age characterised by chronic anovulation, hyperandrogenism and insulin resistance[(2)]. It is a complex disorder where in genetic variants and environmental factors combine and interact to contribute to the patho physiology.

As per WHO criteria, young adults are in the age group of 18 – 24 years[(3)].

Insulin resistance is defined as a state in which greater than normal amount of insulin are required to produce a qualitatively normal response. It is a condition where insulin has less than normal effects in muscle, liver and fat.

- In Adipose Tissue – Increased Hydrolysis of stored Tryglycerides, elevated circulation free fatty acids
- In Muscle – Decreased glucose utilisation
- In Liver – Increased Hepatic Gluconeogenesis Result is an increased blood glucose concentration and compensatory hyper insulinemia[(1)].

In a selected South Indian population, Chennai – the current prevalence of insulin resistance is 17.5 – 18 % (Madras Diabetic Research Foundation).

Overall prevelance of insulin resistance in PCOS ranges between 50% and 75 %.It is a common feature in obese and to a lesser extent in lean woman with PCOS.

Women with PCOS up to 35 % exhibit impaired glucose tolerance and 7 to 10 % meet criteria for Type 2 diabetes mellitus.

Women with Type 2 Diabetes are 6 fold more likely than non diabetic woman of similar age and weight to have PCOS.[(1)]

The discovery of the fact that Insulin resistance plays a central role in the pathogenesis of PCOS is extremely important from clinical point of view.

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Furthermore insulin resistance is associated with an increased risk for various disorders like Type 2 Diabetes mellitus, Hypertension, Dyslipidemia, heart disease, endometrial cancer, recurrent miscarriages and pregnancy complications like GDM, Preeclampsia.[2] [4] [5]

Secondary prevention measures as those that identify and treat asymptomatic persons who have already developed risk factors but in whom the condition is not clinically apparent – early case finding of asymptomatic disease.

This study is aimed at Secondary prevention to identify subjects with insulin resistance at a very early age, couselling regarding the risks, creating awareness in a subset of population so that the natural history of the disease can be altered to maximise the well being of an individual.

Timely intervention could perhaps prevent or at least delay the onset of the above mentioned conditions.

This is a cross sectional survey with nested case control study.

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AIM OF THE STUDY

To measure the prevalence of insulin resistance in young adults with abnormal menstrual pattern.

Long cycles / Scanty periods (Oligo ovulation) was the abnormal menstrual pattern in this study measuring the prevalence of insulin resistance in young adults with Polycystic Ovarian Syndrome.

To correlate the effect of psychological stress on abnormal menstrual bleeding.

REVIEW OF LITERATURE

SECTION A

A HISTORICAL PERSPECTIVE

The name ‘Menstruation’ comes from Latin ‘menses’ meaning month and greek word ‘mene’ meaning moon, with reference to the fact that lunar month is also approximately 28 days long and a complete menstrual cycle usually takes about 28 days.

Length of the menstrual cycle and its variability throughout a woman’s reproductive life are available in the works of Matsumoto et al. In 1962, Treolar et al, in 1967 Chiazze et al,in 1968 and Vollman et al. in 1977, investigated menstrual bleeding patterns.[6] [7]

Among the large population based studies cited above Matsumoto et al. in 1962 has reported on length of the bleeding episode in a cycle. Long Menstrual periods (more than eight days) were associated with ovarian dysfunction, being more common in anovulatory cycles and in cycles with an inadequate luteal phase.[8]

In 1935, Irving F. Stein and Michael L Leventhal first described a symptom complex associated with ovulation. In their studies, Stein and Leventhal described association between the presence of bilateral

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polycystic ovaries and signs of amenorrhea, oligomenorrhea,obesity and hirsutism .These signs were strictly adhered in diagnosis of what was known as Stein-Leventhal Syndrome.[9] [10]

Even earlier the association between a disorder of carbohydrate metabolism and hyperandrogenism was first described in 1921 by Archard and Thiers and was called ‘diabetes of bearded women’.

Review related to genetic basis of PCOS

Kierland et al in 1947 reported the skin lesion, acanthosis nigricans to occur frequently in women with hyperandrogenism and diabetes mellitus.[11]

Cooper et al., in their study, described that the incidence of oligomenorrhea and polycystic ovaries is increased in first-degree relatives of PCOS patients when compared with the controls.[12] [13]

Givens et al. reported a series of family-based studies, using a diagnostic criteria consisting of hirsutism, oligomenorrhea, and enlarged ovaries. They found familial aggregation of hyperandrogenic and metabolic disorders.

Hague et al. used high-resolution ultrasonography to identify polycystic ovaries in 61 women with obesity, menstrual disturbances, hyperandrogenism and infertility and in their first-degree female relatives

Lunde et al. Studied 132 Norwegian women families identified on the basis of an ovarian wedge resection, who had two or more of the following symptoms: menstrual irregularity, hirsutism, infertility.[12]

Brown et al in 1968 found that there was an association between insulin-resistant diabetes mellitus and genetic basis as suggested by reports of affected sisters.

Kahsar-Miller et al used NICHD criteria for the diagnosis of PCOS and reported rates of PCOS in sisters and mothers of patients with PCOS as 32% and 24%, respectively.[12]

Legro et al. showed that 24% had increased *T* and DHEAS values with regular menstrual cycles, 22% of reproductive aged sisters of women with PCOS met the diagnostic criteria of PCOS.

Quantitative phenotypes related to glucose homeostasis and hyperandrogenemia are shown to be heritable in PCOS. Evidence for heritability of metabolic phenotypes such as insulin resistance and beta cell function was reported in family studies of PCOS.

Norman et al reported that increased insulin levels were common among first-degree relatives after studying the families of five patients with PCOS.[11]

Colilla et al. noticed that there was a heritable component of beta cell dysfunction in families of women with PCOS.[14]

Review related to poly cystic ovarian syndrome.

PCOS affects 7% of women from all races and nationalities .Polycystic ovary syndrome (PCOS) is also known as Stein Leventhal Syndrome. It was first described in 1935.

One of the most common symptoms of PCOS is irregular periods. Polycystic ovary syndrome (PCOS) becomes symptomatic during adolescence and affects 5% of reproductive-age women.[9]

PCOS is a heterogeneous syndrome of unexplained oligo-anovulation and chronic hyperandrogenism, with polycystic ovary being an alternative diagnostic criterion.[15] [16]

About 50 % of cases lack some classic features of obesity, polycystic ovaries ,menstrual irregularity, hirsutism.

Minerva Pediatr in 2010 showed that Polycystic Ovary syndrome (PCOS) is a complex disorder primarily involving ovarian hyperandrogenism in females and linked with insulin resistance in most of the cases.(11)

Review Related To Prevalence

Pembe AB, Abeid MS in 2009 conducted a study to determine prevalence of polycystic ovaries and associated biochemical and clinical features among women with infertility attending gynaecological outpatients department at Muhimbili National Hospital in Tanzania.

Acne and oligomenorrhoea were significantly higher in group of women with PCO than among women with normal ovaries. The mean hirsutism score was higher in women with PCO than in women with normal ovaries, though it was not significant (5.1 +/-2.7 vs 4+/-2.4, P<0.057).

Using the Rotterdam criteria 32 women were diagnosed to have polycystic ovary syndrome. Among these women 25 had PCO, 24 had oligoanovulation and signs, and 18 had hirsutism. Among 68 women with no PCOS, 7 of them had polycystic ovaries, 15 of them had signs and oligoanovulation and 6 of them had hirsutism.

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In conclusion they showed polycystic ovaries are common among women with infertility, however not necessarily associated with polycystic ovary syndrome.[11] [13] [17]

Koivunen R. (1999) conducted a study on prevalence of polycystic ovaries in healthy women. The prevalence of polycystic ovaries varied with age. The findings were most common in women with 35 years of age or younger than in those 36 years aged or older.[11] [18]

Enhrman DA, Barnes RB, Cavagham MK in the year 1999 studied prevalence of impaired glucose tolerance and diabetes in women with polycystic ovary syndrome and found that obese women with polycystic ovary syndrome have the highest risk of glucose intolerance.[11]

The study showed hyperandrogenemia may have a role in the development of glucose intolerance or may be a marker of insulin resistance, 35 % have impaired glucose tolerance and 10% before the age of 40 became diabetic.[19]

Conversion to NIDDM from impaired glucose tolerance appears to be accelerated in women with PCOS.

Review related to quality of life

Moran L, et.al in the year 2010 assessed the psychological features in young women with and without PCOS.

Women with PCOS demonstrated greater anxiety and depression along with worsened quality of life than women without PCOS .It was an observational, cross sectionalstudy.[11]

Judy Griffin Mc Cook, et al conducted a study on quality of life in women with polycystic ovarian syndrome.

The results of this study indicated that women with PCOS had problems in the area of weight, followed by menstrual problems and infertility.This was a cross sectional study.[11] [20]

These concerns directly reflected the objective life experiences. Women with PCOS thereby clearly need support and education regarding the effect of their quality of life.

Maria E el. al conducted a study on female adolescents to determine whether clinically observed or self –perceived severity of illness affect their HRQL (Health related quality life).

They concluded that adolescents with PCOS experienced lower HRQL compared with healthy adolescents.

This study suggested the need to develop interventions to reduce the distress that patients with PCOS may face to adolescents and young adult.

Susanne Hahn, el. al showed that PCOS patients had significant reductions in quality of life, decreased sexual satisfaction and increased psychological disturbances than healthy controls.[21]

Review related to risk factors

Angela Kerchner, B.A., et .al in 2009, conducted a prospective longitudinal study and concluded that there is a significant risk of mood disorders in women with polycystic ovarian syndrome.

The high rate of depression and other mood disorders are present in young women with PCOS.[22]

Rosenfield et al, conducted a study on polycystic ovary syndrome and found to arise as a complex trait with both heritable and nonheritable factors.

Polygenic influences accounted for about 70% of the variance in pathogenesis.[11] [23]

Fertile steril in 2009 conducted a prospective longitudinal study and stated that there is a significant risk for mood disorders in women with polycystic ovarian syndrome. They found that routine screening and identifying mental health disorders is important because there is a high conversion risk for depression.

Want Y, et.al says that family history of diabetes mellitus has most of the effect on the clinical phenotype in women with PCOS.[20]

Wilson et al. in 1984 and Osofsky and Fisher in 1967, Metcalf and Mackenzie in 1980, support Drew's theory of an association between stress and amenorrhea. However more rigorous studies are needed.

Metcalf and Mackenzie in their study have shown that young women who live at home ovulate more frequently than do women who live in flats or hostels. Five to eight years after menarche, 72 percent of girls living at home ovulated, compared with only 40 percent of women who lived away from home

Review related to measuring Insulin Resistance

Legro Et al. In 1998 showed Fasting Glucose to Insulin ratio is a useful sensitivity index to measure insulin resistance in PCOS.[24]

Vuguin et al. 86 showed Fasting Glucose to Insulin ratio is a useful sensitivity index to measure insulin resistance in PCO.[25]

American Diabetic Association (ADA) has recommended to screen for insulin resistance with fasting levels of insulin and glucose in PCOS.Fasting levels have been shown to have good correlation with more intensive measures of insulin action in a PCOS population (glucose/insulin ratio < 4.5 consistent with decreased insulin sensitivity).

Golbahar et al in 2012 used fasting glucose insulin ratio to determine the sensitive and specifc markers for insulin resistance.[26]

Kauffman et al showed that a fasting glucose insulin ratio less than 7.5 is a sensitive measure of insulin resistance in obese and non obese patients with PCOS.[27]

Review Related to Stress and Menstrual Cycle

Although psychological stress is generally acknowledged to affect menstruation and is often considered the principal cause of menstrual

dysfunction, scientific investigation of the association between stress and menstrual function is actually quite limited.

Drew in 1961 concluded that the amenorrhea observed in these studies was attributable to psychological stress characterized by separation from home and family, hopelessness and threat to the individual.[11]

SECTION B

Accepted norms for Menstrual

Frequency – 24 to 35 days

Regularity + or _ 5 days variation

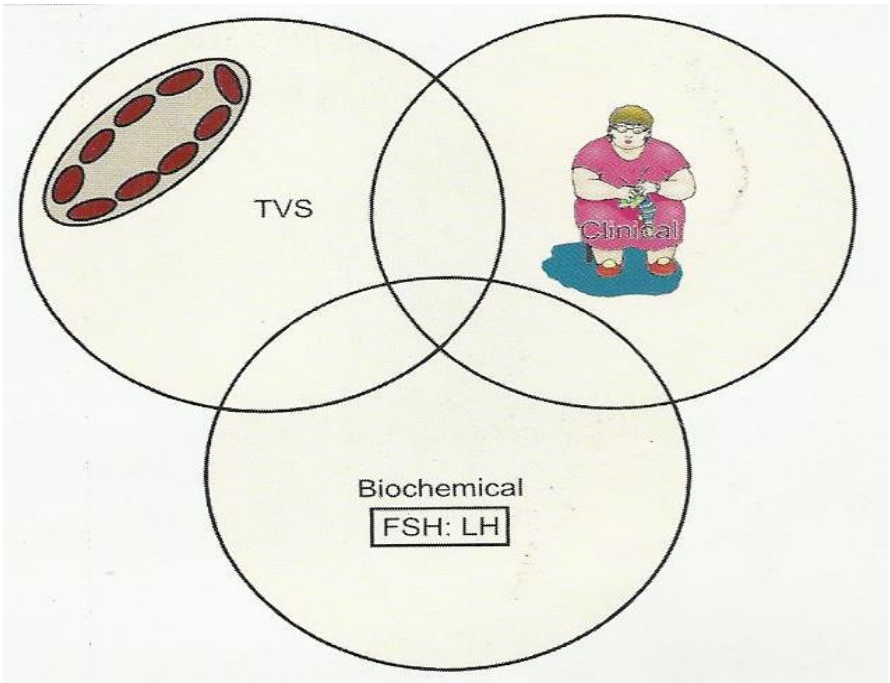
Duration – 2 to 7 days

Oligomenorrhoea – In frequent menses occurring at intervals > 35 days.[1]

PCOS an ill defined heterogenous condition with a complex pathophysiology, is one of the commonest endocrine metabolic disorders. PCOS is not only a fertility disorder, but an important general health problem where the central etiological factor is most likely insulin resistance [28]. Defining this syndrome has always remained a challenge for clinicians due to the wide variety of presentations.

National Institute of Child Health and Human Development (NICHD) (1990)	European Society for Human Reproduction and Embryology (ESHRE) and American Society for Reproductive Medicine (ASRM) – Rotterdam – 2003	Androgen Excess And PCOS Society (AE-PCOS) 2006
Hyperandrogenism and / or Hyperandrogenemia	Clinical or biochemical signs of hyper androgenism	Hyperandrogenism (Hirsutism and / or Hyperandrogenemia
Menstrual Dysfunction	Oligo / Anovulation	Ovarian Dysfunction (Oligo / Anovulation and / or Polycystic Ovaries)
Exclusion of other known disorders having a similar clinical presentation	Polycystic Ovaries (As identified by ultrasonography) also excluding other androgen excess disorders	Exclusion of other androgen excess or related disorders

(1)



Diagnosis of the PCO

Insulin resistance

Since an initial report in 1980 of the association between PCOS and Hyperinsulinemia, it is apparent that women with PCOS are insulin resistant in relation to weight match control.

Hyper insulinemia plays a role in the etiopathogenesis of hyperandrogenism in women with PCOS by :

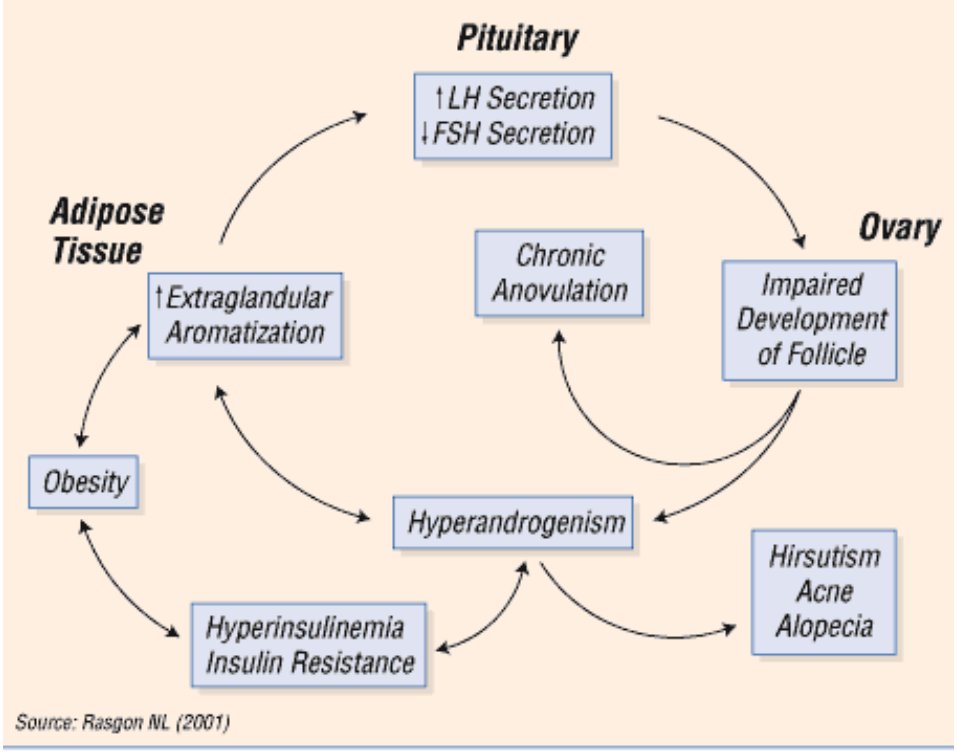
1, increasing the ovarian androgen production

2, Decreasing the serum Sex hormone binding globulin concentration[29]

Cytochrome P 450 C, 17 alpha is a bifunctional enzyme that has both 17 alpha hydroxylase and 17,20 lyase activity and is key in the bio synthesis of ovarian androgens.[30]

Nester et al hypothesized that hyperinsulinemia stimulates ovarian P 450 C and 17 alpha activity in women with PCOS and amelioration of insulin resistance in these women would return the activity of enzyme towards normal and was also accompanied by a decline in serum free testosterone concentration.[31]

Pathophysiology of Polycystic Ovary Syndrome



INSULIN ACTION:

Actions of insulin are mediated by two distinct intracellular pathways and its receptor.

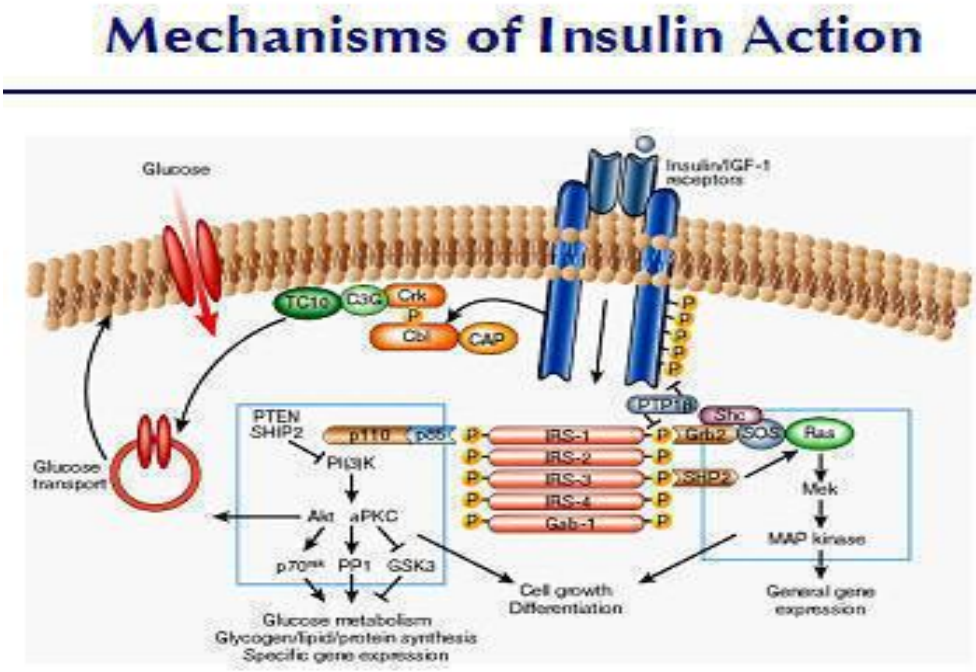
- Phosphatidyl-inositol 3- kinase (PI-3K) pathway – mediates the metabolic effects of insulin.
- Mitogen-activated protein kinase (MAPK) pathway - mediates the proliferative actions of insulin.

Insulin binding to its receptor results in tyrosine phosphorylation of the receptor and protein substrates. This in turn bind and activate PI-3K and Akt serially.

Akt is an effector molecule which plays major part in transduction of signal for regulation of glucose and metabolism.Activation of Akt potentiates the translocation of (GLUT 4) or Glucose Transporter 4 from inside the cell to the plasma membrane, hence increasing glucose uptake.

Insulin inhibition of gluconeogenesis and glycogenolysis is mediated by other effector molecules. It also stimulates lipid synthesis and inhibits lipid catabolism. In PCOS there is a selective increase in insulin activation of the MAPK pathway and resistance in PI-3K mediated pathway.

This observation shows how insulin actions can be inhibited and enhanced at the same time by different pathways. This also explains how insulin stimulates hyperandrogenism in insulin resistant women.Resistance to insulin and hyperinsulin secretion plays an important part in the pathophysiology of PCOS.(1) (32)



HOW TO TEST:

1. The gold standard to measure sensitivity of insulin is known as hyperinsulinemic euglycemic clamp.One fixed intravenous infusion (IV) of insulin and simultaneously another IV glucose infusion is started. Varying the rate of glucose infusion , a steady state plasma glucose level within the normal fasting range is achieved.

The lower the rate of glucose infusion at a steady state, the greater the degree of resistance of insulin.[1]

2. Fasting serum insulin concentration:

In women with PCOS value more than 20 – 30 micro unit / ml suggest insulin resistance.[1]

3. Fasting glucose insulin ratio:

A ratio less than 4.5 is a good measure for specificity and sensitivity of insulin resistance.[1]

4. Homeostatic model assessment of insulin resistance(HOMA-IR):

Calculation is made by dividing the product of fasting glucose and insulin level by a constant. Value greater than 3.2 – 3.9 suggest resistance to insulin.[1] [33]

5. Quantitative insulin sensitivity check index (QUICKI):

Expressed logarithmically, as the inverse of sum of fasting glucose and insulin level.

Values greater than 0.33 indicates insulin resistance.[1][34]

6. Oral Glucose Tolerance Test:

It involves measurement of plasma insulin and glucose over a period of 2 hours after a 75gm or 100gm glucose load.[1]

Interpretation	2- Hour Glucose	2-Hour Insulin
Normal	<140 mg/dl	
Impaired Glucose Tolerance	140-199 mg/dl	
Diabetes Mellitus	_>200 mg/dl	
Normal		<80-100uU/ml
Insulin Resistance		>80-100uU/ml
Severe Insulin Resistance		>300uU/ml

(1)

Women with PCOS generally exhibit increased serum LH concentrations, increased LH to FH ratios, low – normal FSH levels[35]

Increase in serum LH level are seen in 40 % of women and it is due to :

1, Increases in LH amplitude , pulse frequency which is in turn due to increase in GnRH pulse frequency.

2, Abnormal LH secretory dynamics[36]

Decrease in FSH levels are due to

1, Negative feedback of chronically elevated estrone concentration

2, Increase in GnRH pulse frequency

3, Increased levels of Inhibin B[1]

Diagnostic Criteria in PCOS

A, Clinical

B, Biochemical

C, Ultrasound

Clinical[39]

Menstrual History – Amenorrhoea and oligomenorrhoea are common complaints.

20 % of patients with PCOS had a history of regular cycles in 10 years post menarche which later becomes irregular.

28 % of all women with PCOS report irregular menses.[40]

Kiddy reported a 72 % incidence of menstrual disturbance in lean PCOS.[2] [41]

History of weight gain and calculation of Body mass Index[42]

$$BMI = \frac{\text{Weight in kilogram}}{\text{Height in } m^2}$$

Underweight	< 19.9 kg / m^2
Normal	20-24.9 kg / m^2
Overweight	25 – 29.9 kg / m^2
Obese	>30 kg / m^2 (43)

Evidence of Androgen Excess

Acne : About 30 % of patients of PCOS will have acne.[44] [45]

Hirsutism: Hirsutism is defined as growth of terminal hair in a woman in the same pattern and sequence as seen in the normal male.

It is a change in the quality , size, length of hair as well as degree of pigmentation and not the number of hairs. About 9 % of young female population is considered to be hirsute and in about 60 – 70 % of cases i is due to PCOS.[46]

Acanthosis Nigricans.[47]

Acanthosis Nigricans is a mucocutaneous eruption characterised by hyperkeratosis, papillomatosis and increased pigmentation. It is seen in 5 % of women with PCOS.

The Lesions have a velvety contour and are commonly seen in the axilla, nape of the neck, under the breasts or in the flexures. It is a sign of insulin resistance.[48] [49]

The term “HAIR-AN” syndrome is used to describe HyperAndrogenism, Insulin Resistance and Acanthosis Nigricans.[2]

Biochemical Test.[50]

The goal is to assess.

1, the severity and source of androgen excess.

2, rule out other sources of hyperandrogenism such as adrenal tumour, cushings disease and congenital adrenal hyperplacia.

3, To screen for associated endocrine disturbances in PCOS.

Endocrine disturbances

FSH and LH :

LH : FSH Ratio is greater than 3 in 40 % of patients[43]

Thyroid stimulating Hormone and Free Thyroxine :

Thyroid dysfunction may cause amenorrhoea and Hirsutism

TFT may be abnormal in 5 % of patients with PCOS[51]

Prolactin:

15 % of patients with PCOS have increase prolactin levels. This may be due to the syndrome per se or may be an coincidental finding. It is necessary to rule out other causes of hyper prolactinemia in these patients.[52]

Total and Free Testetrone:

Elevated in patients with PCOS

DHEAS: (Dehydro Epi Androsterone Sulphate)

Mainly indicates adrenal contribution to androgens[53] [54]

Ultrasound[55] [56]

Sonographic Criteria for PCO (Adam)

1. Multiple (> 10) small (2-8 mm) peripheral cysts
2. A dense core of stroma
3. Enlarged ovaries [> 8 ml (8 – 14 mm]

Sonographic Criteria for PCO

Ardaens Sonographic Criteria (1991)[55]

In 1991 Ardaens had also described criteria for the ultrasound diagnosis of polycystic ovaries, though they are less used now due to the low sensitivity.

Ardaens / Robert Criteria

External Morphological Signs

1, Increased Ovarian area and volume

2, Increased roundness index (Ovarian width / length ratio < 1)

3, Decreased uterine width / ovarian length ratio (U / O)

Internal Morphological Signs

1, Number of small echoless regions < 10 mm in size per ovary (micro cysts)

2, Peripheral repartition of microcysts

3, Increased echogenicity of the ovarian stroma

4, Increased surface of the ovarian stroma on a cross- sectional cut (computerized measure)

Differential Diagnosis of Anovulatory Disorders and Associated Serum Laboratory Findings

	FSH	LH	Prolactin	Tesetetrone
Extreme exertion or rapid weight changes	Normal	Normal	Normal	Normal
Premature ovarian failure	Significantly elevated	Moderately elevated	Normal	Normal
Pituitary Adenoma	Mildly reduced	Mildly reduced	Moderately elevated	Normal
Progestational Agents	Mildly reduced	Mildly reduced	Normal	Normal
Hyper/ Hypothyroidism	Normal	Normal	Normal to mildly elevated	Normal
PCOS	Normal to mildly reduced	Moderately elevated	Normal to mildly elevated	Normal to moderately elevated
Congenital Adrenal Hyperplasia	Normal	Normal	Normal	Normal to mildly elevated

Health Consequences[57] [58]

PCOS and the Risk of Diabetes[59] [60]

There is an increased risk of developing impaired glucose tolerance and frank diabetes mellitus in PCOS.

This association was first recognised by Achard and Theirs in 1921. Since then Dunaif et al clearly demonstrated much greater prevelance of non insulin dependent diabetes mellitus in PCOS when compared to age and weight matched controls.

This finding was seen initially in obese patients , though later studies have shown a higher risk in lean women (BMI <less than 27 kg / m 2).

Ehrmann et al found diabetes in 10 % and IGT in 35 % of women in PCOS.

Insulin resistance and metabolic abnormalities worsen with age and hence most cases of diabetes are seen in middle age.

Insulin resistance combined with elevated androgen concentration seen in PCOS increases the risk for diabetes.[61] [62]

PCOS and Cardio Vascular Diseases.[63] [64]

Dahlgren et al found 4 to 11 fold increased risk of coronary heart disease in PCOS.

These patients were having hyperlipidemia with raised low density LDL and Ap01 level. Risk of coronary artery calcification was higher and atherosclerosis was higher. This dyslipidemia is seen in both lean and obese PCOS.[65] [66] [67]

PCOS and Recurrent Miscarriages

Rai R et al reported incidence of 40.7 % of recurrent miscarriages in PCOS due to elevated levels of LH and Androgens.[68]

PCOS in Pregnancy

PCOS has been linked to development of gestational diabetes, pregnancy induced hypertension, preeclampsia and premature delivery.[69]

Insulin resistance is associated with hypertension and hypertensive disorders of pregnancy.

Prevelance of Preeclampsia and PIH was found to be higher in PCOS patients by Urman et al and Fridstom et al.

PCOS and Cancer

There is an unopposed estrogen exposure due to anovulatory amenorrhea or oligomenorrhea.

This Hyperestrogenic stage can result in increased mitotic activity in responsive tissues such as endometrium and breast.

Due to this stimulus certain dysplastic changes may occur that results in endometrial hyperplasia, endometrial cancer or breast neoplasia.[70] [42]

Intervention

There are 3 major lines of intervention in patients with PCOS. [71][72] [73]

1, Lifestyle changes – Healthy diet and regular excercise.

2, Medical Management – Medications to induce ovulations to control the increased insulin resistance to induce endometrial shedding and to control symptoms of hyperandrogenism.

3, Surgical Treatment – Laparoscopic ovarian diathermy / Laser drilling can restore ovulatory cycles.

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MATERIALS AND METHODS

This is a cross sectional survey with nested case control study carried out at ESI Medical college and PGIMSR and as a Health Camp initiative 11 visits at MEPZ, Tambaram , to screen young adults working in a export factory , who were ESI beneficiaries.

Time period:

August 2012 to August 2013

Study group:

18 – 24 years

Sample Size : 223

To improve the accuracy of the study and due to availability of study cases, 129 young adults with long cycles / scanty periods were enrolled in the test group and 93 young adults who had regular cycles were enrolled in the control group.

Inclusion Criteria :

- Young women in the age group of 18 – 24, irrespective of the marital status.
- Normal menstrual pattern since menarche.
- Any menstrual deviation in duration, cycle, amount.

Exclusion Criteria:

- Age group less than 18 and more than 24
- Amenorrhoea due to pregnancy
- Primay amenorrhoea
- Young women on oral contraceptive pills
- Ultrasound detection of fibroids, ovarian tumours

After obtaining informed consent, a history on their menstrual pattern was elicited. Those who had long cycles / scanty periods and those with regular cycles were enrolled in the study.

All the subjects attended ESIPGIMSR, where a detailed clinical examination and relevant history including Questionnare for Perceived Stress Scale Score was recorded.

They were subjected to blood investigations in the Biochemistry Laboratory of our hospital. After overnight fasting blood was collected for Fasting glucose, Fasting Insulin, Thyroid Function Tests, Serum Prolactin, Serum FSH, Serum LH, Serum Testosterone, DHEAS, Lipid profile. 75 gm glucose was given and 2 hours blood sample was collected for Glucose and Insulin levels.

Ultrasound pelvis was done by the hospital radiologist.

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STATISTICAL ANALYSIS:

Data analysis was done by a Statistician, with the help of computer using Epidemiological Information Package (EPI 2010) developed by Centre for Disease Control, Atlanta.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables and Yate's chi square test for qualitative variables.

A 'p' value less than 0.05 is taken to denote significant relationship.

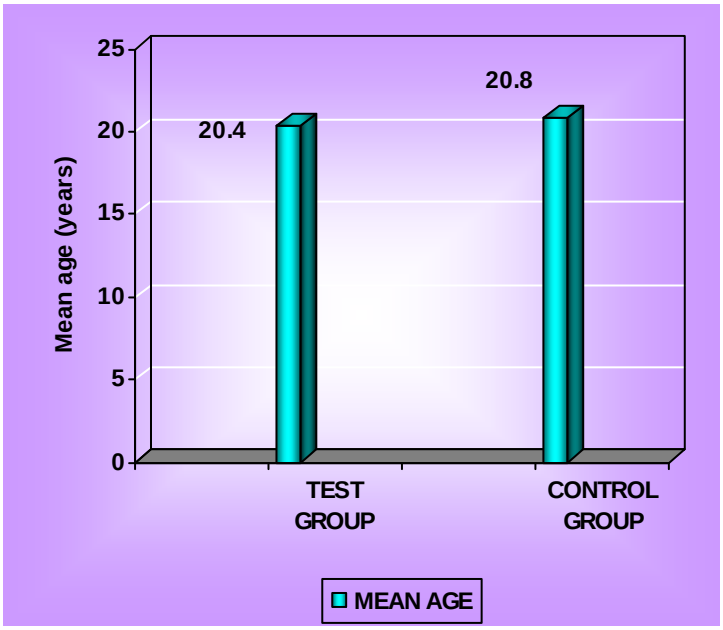
OBSERVATION AND RESULTS

Test Group :

Women with long cycles/ scanty periods – 129 cases

Control Group :

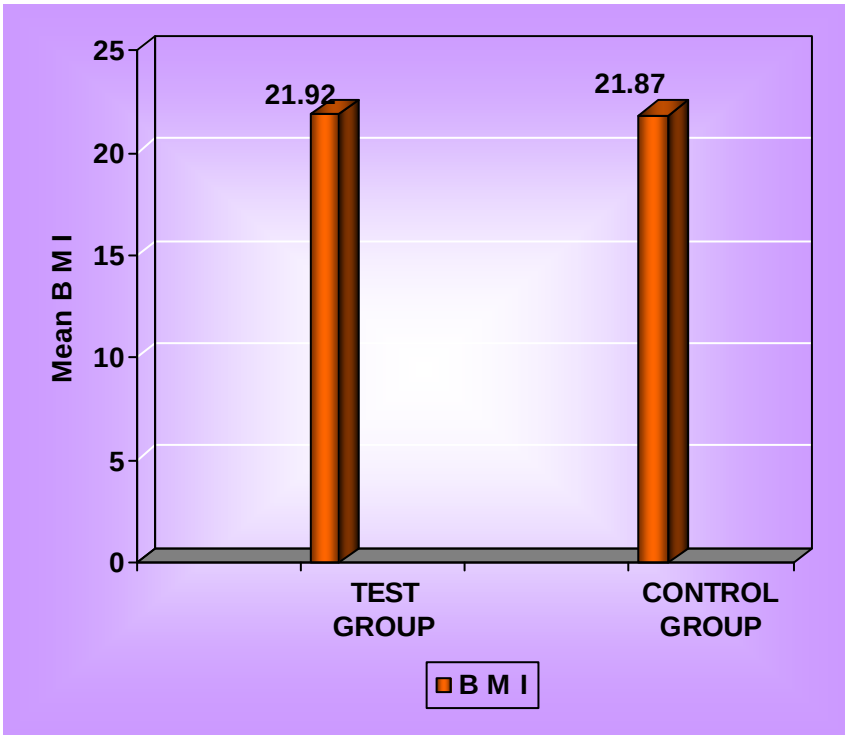
Women with normal menstrual cycles – 93 cases Age distribution



Group	Age in years	
	Mean	SD
Test group	20.4	1.8
Control group	20.8	3.1
'p'	0.6947 Not significant	

Among the women with long cycles / scanty periods and women with normal cycles there is no statistically significant difference in their mean age.

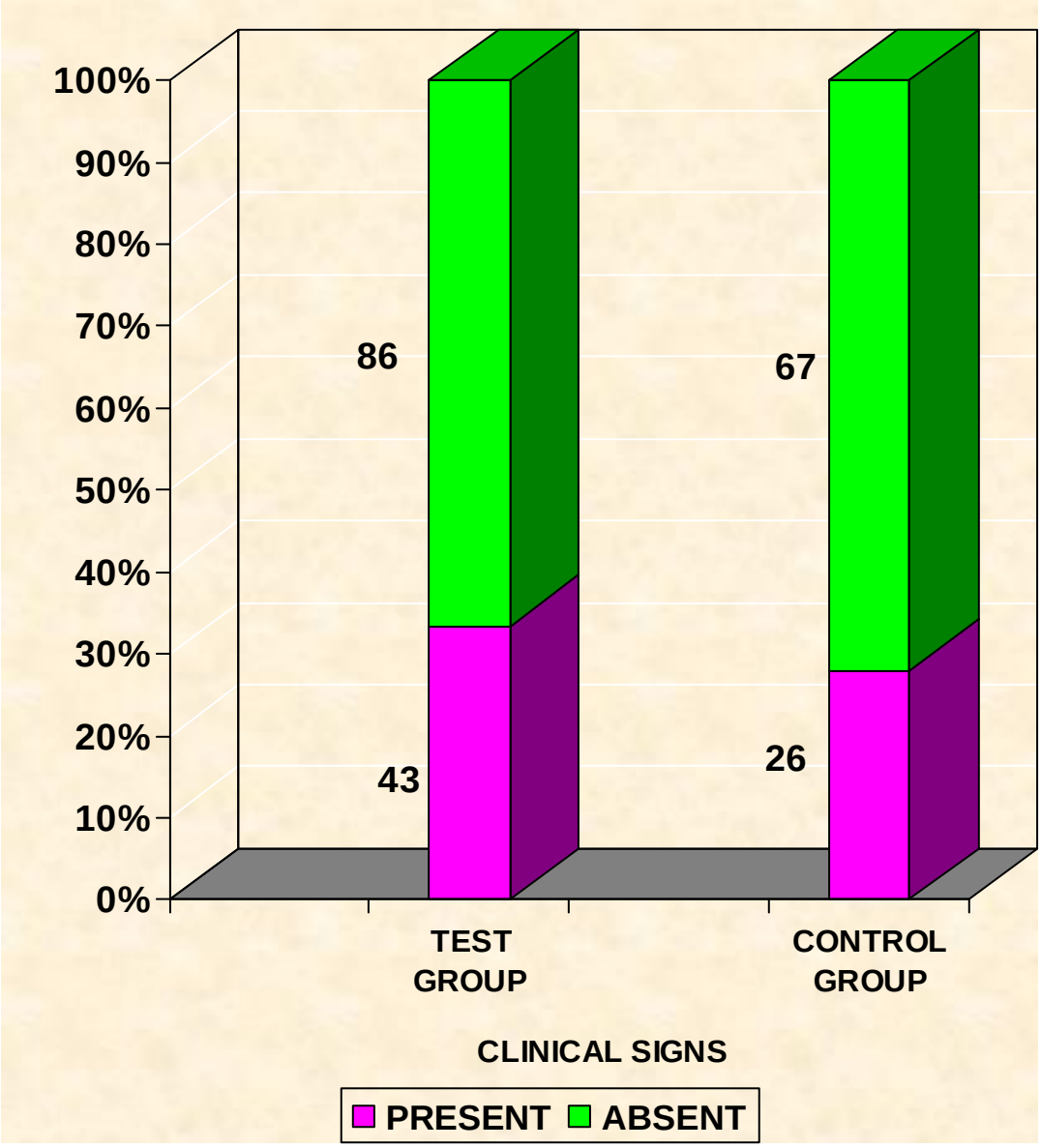
BODY MASS INDEX



Group	BMI					
	Mean	SD	Normal		Abnormal	
			No	%	No	%
Test group	21.92	2.22	115	89.1	14	10.9
Control group	21.87	2.11	84	90.3	9	9.7
'p'	0.7563 Not significant					

Body mass index did not have any statistically significant difference between the test group or control group

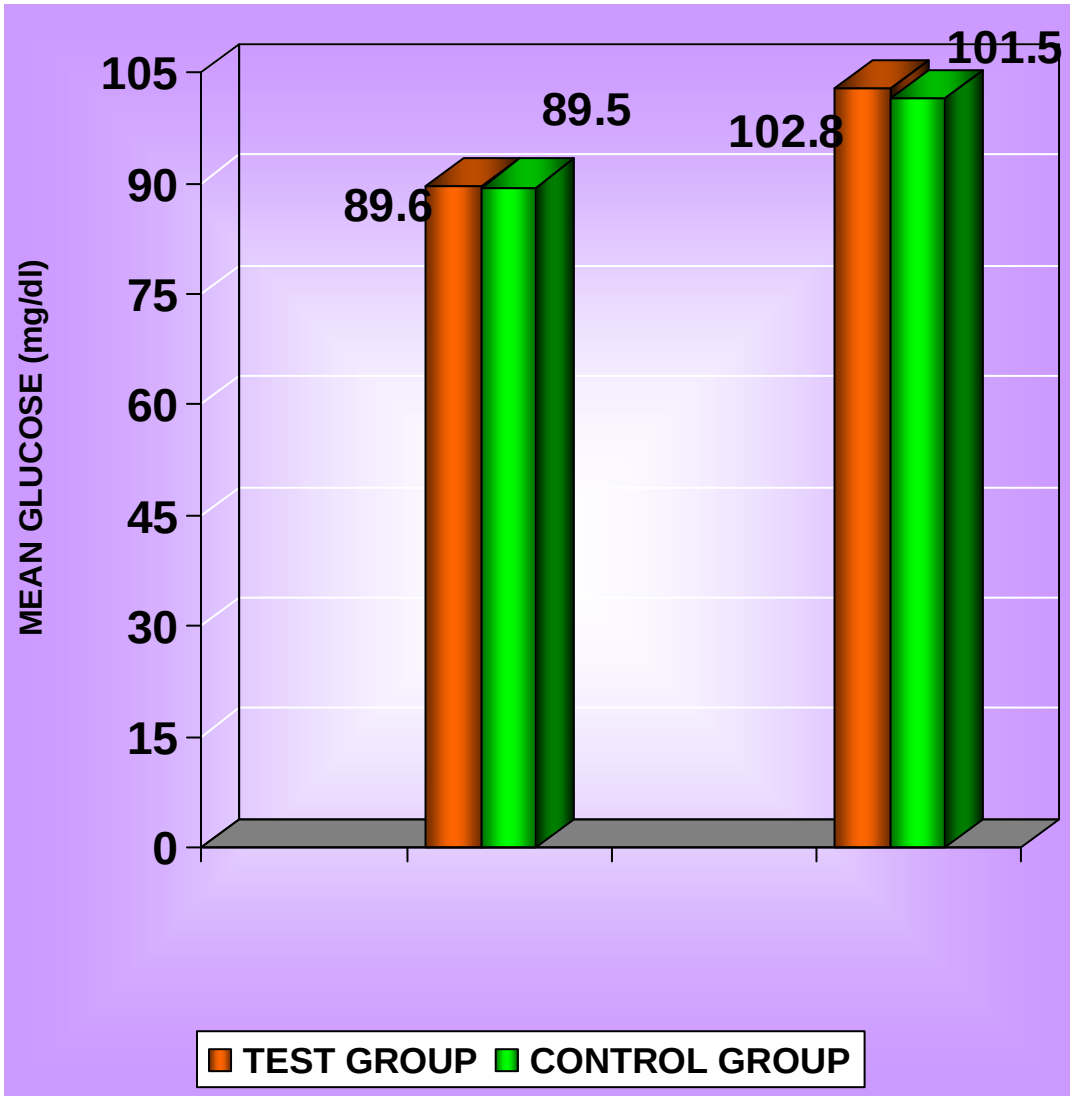
CLINICAL SIGNS



Clinical signs	**Test group (n=129)**		**Control group (n=93)**	
	No	**%**	**No**	**%**
Acne	43	33.3	26	28.0
Hirsutism	1	0.8	5	5.4
Acenthosis nigricans	8	6.2	9	9.7
Total cases with clinical signs	43*	35.2	26*	28.0
Cases without clinical signs	86	66.7	67	72.0
'p'	0.4796 Not significant			

Clinical signs are present more in women with long cycles than in normal cycles. There were 43 women in the test group with clinical signs and 26 women in the control group. The p value was 0.4796 which was not statistically significant.

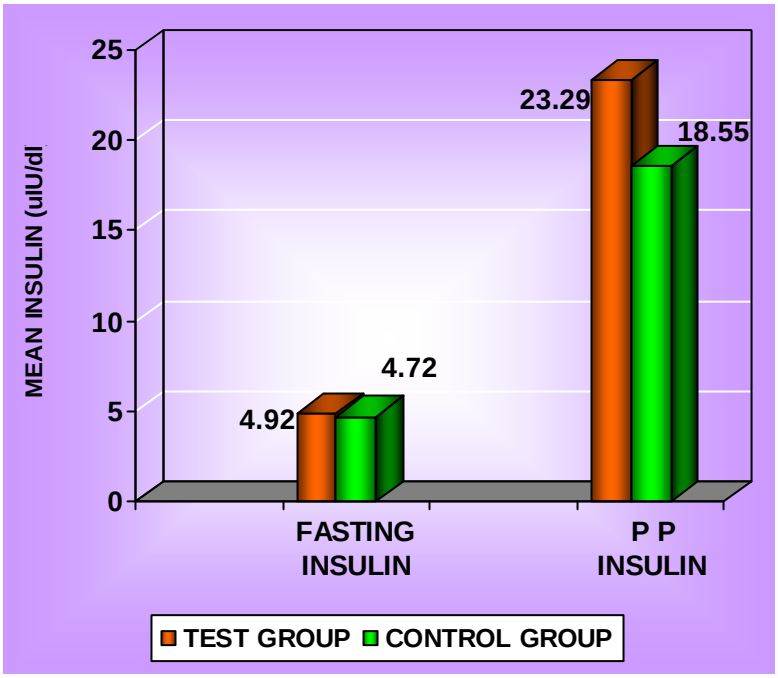
GLUCOSE LEVELS



Glucose	**Test group**		**Control group**		**'p'**
	Mean	**SD**	**Mean**	**SD**	
Fasting glucose	89.6	11.1	89.5	9.5	0.7227 Not significant
Post prandial glucose	102.8	18.3	101.5	25.3	0.2744 Not significant

The mean fasting glucose level in the test group is 89.6 mg/dl, and in the control group is 89.5. The 'p' value is 0.7227 which is not significant. Similarly the mean post prandial blood glucose between the test and control group shows no statistical significance.

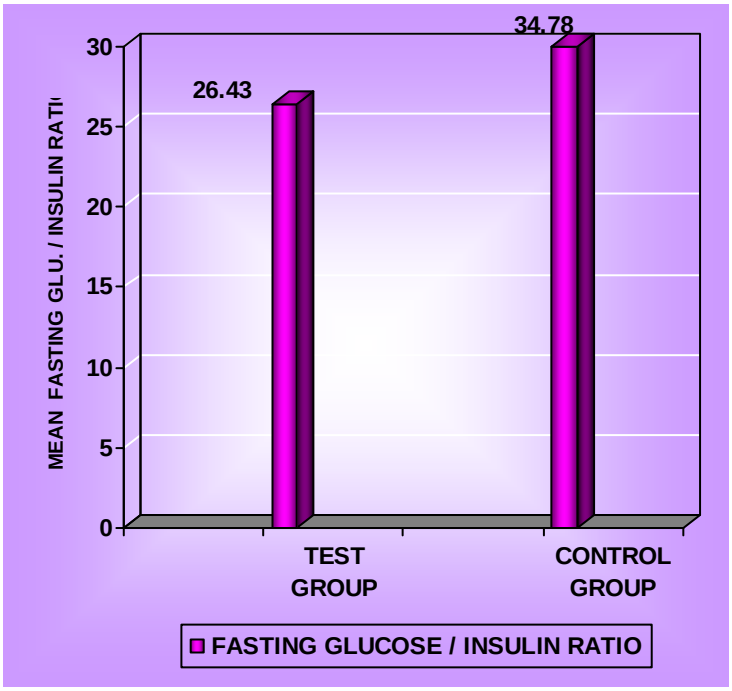
INSULIN LEVELS



Insulin	Test group		Control group		'p'
	Mean	SD	Mean	SD	
Fasting	4.92	3.56	4.72	3.49	0.4136 Not significant
Post prandial glucose	23.29	29.84	18.55	25.57	0.2112 Not significant

There is no statistically significant difference between the mean fasting and post prandial insulin levels among the test group and control group.

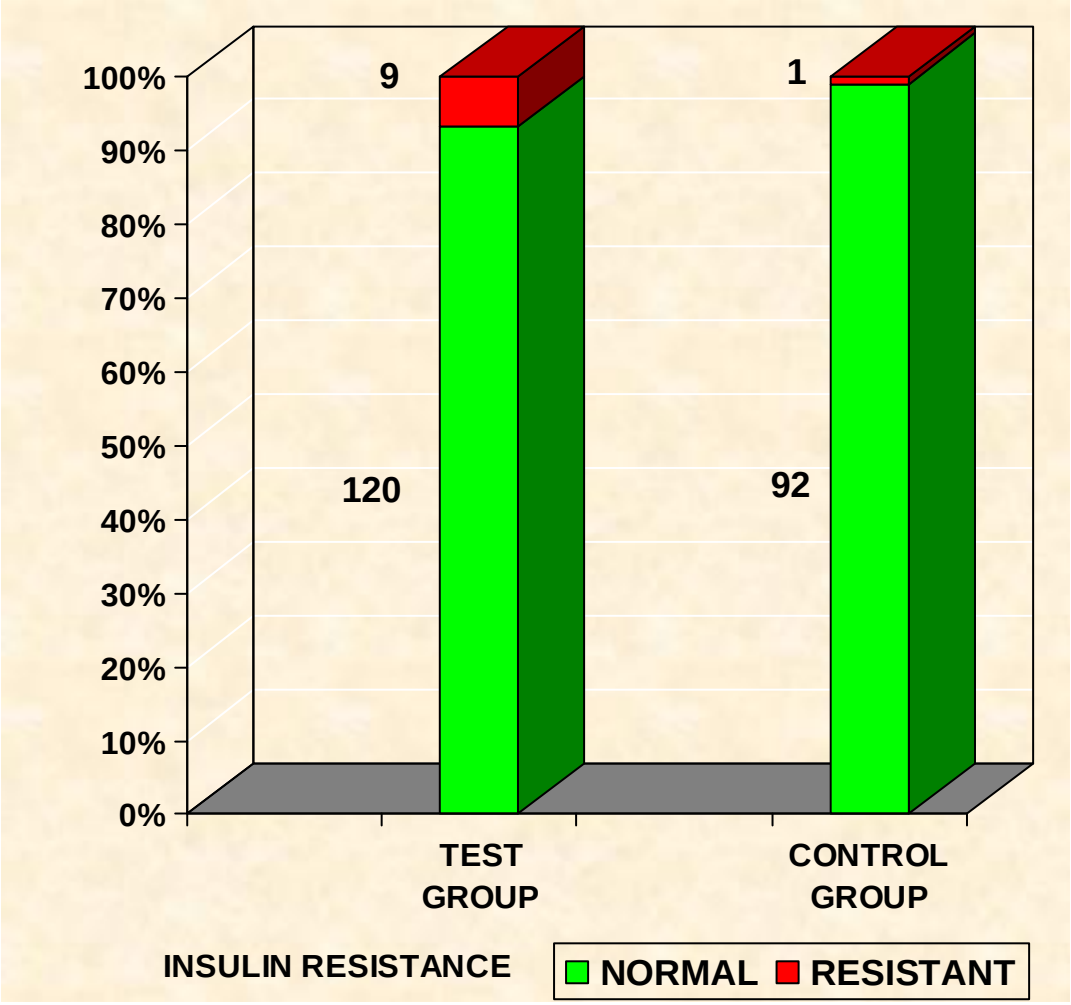
FASTING GLUCOSE / INSULIN RATIO



Fasting glucose insulin ratio	Test group	Control group
Mean	26.43	34.78
SD	9.7	6.4
'p'	0.0346 Significant	

Women with long cycles / scanty periods have a mean fasting glucose / insulin ratio of 26.43 and women in control group have a mean fasting glucose / insulin ratio of 34.78. The 'p' value is 0.0346.This difference is statistically significant.

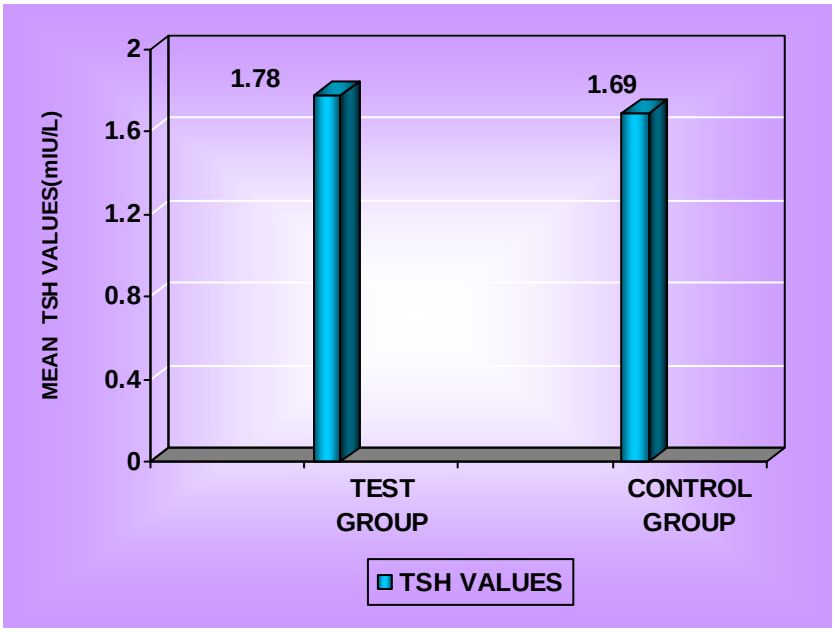
INSULIN RESISTANCE



Group	**Insulin resistance**			
	Normal		**Resistant**	
	No	**%**	**No**	**%**
Test group	120	93	9	7
Control group	92	98.9	1	1.1
'p'	**0.033** **Significant**			

Out of 129 in the test group 9 were insulin resistant with fasting glucose / insulin ratio less than 4.5. Out of 93 in the control group one person was insulin resistant. The 'p' value is 0.033 which is statistically significant as p value is less than 0.05.

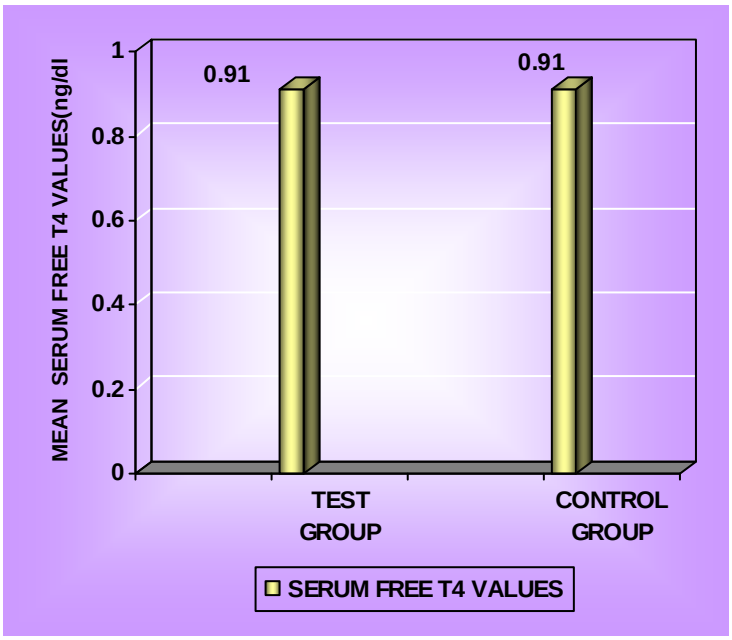
TSH VALUES



Group	TSH values (mIU/L)					
	Mean	SD	Normal		Abnormal	
			No	%	No	%
Test group	1.78	1.58	118	91.5	11	8.5
Control	1.69	2.35	88	94.6	5	5.4
'p'	0.3296 Not significant					

8.5 % in the test group have high TSH values and 5.4 % in the control group.

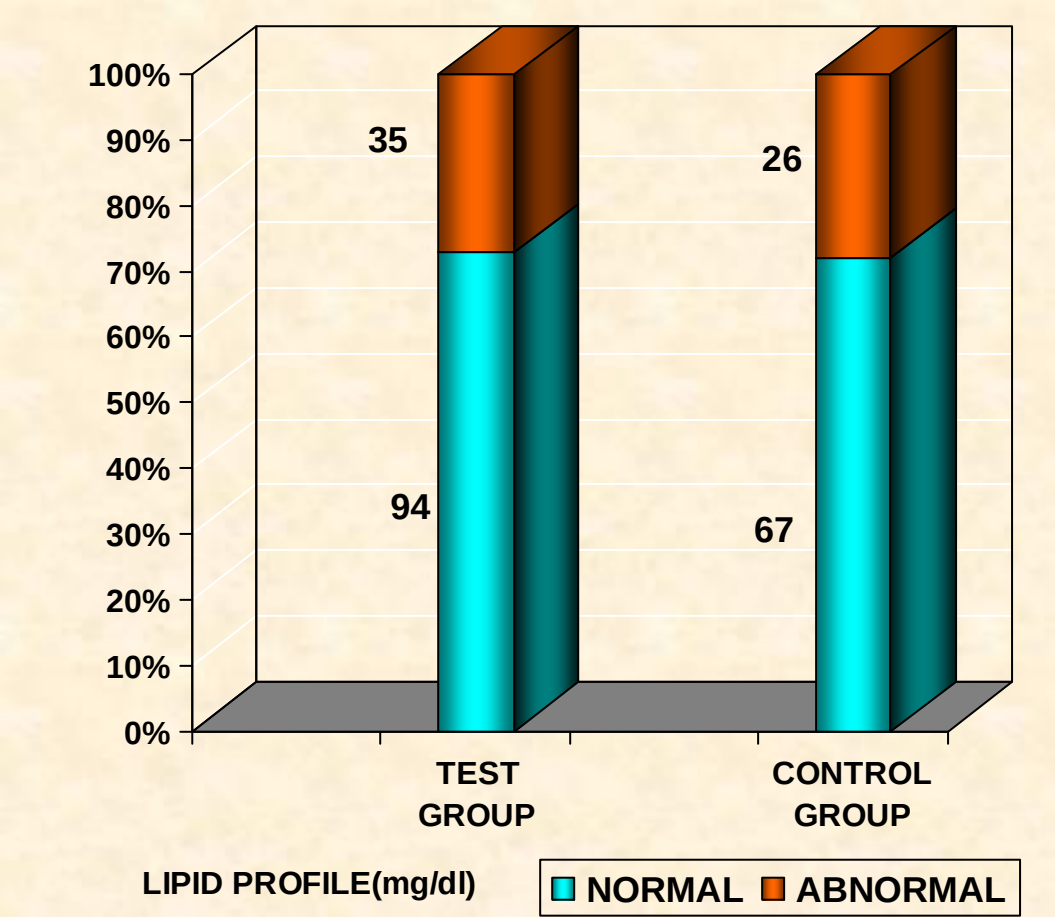
SERUM FREE T4 VALUES



Group	Serum free T4 (mg/dl)	
	Mean	**SD**
Test group	0.91	0.19
Control group	0.91	0.12
'p'	0.2642 Not significant	

Among the women with long cycles and scanty periods and those with normal cycles there is no statistically significant difference in serum T4 values.

LIPID PROFILE



Lipid Profile (mg/dl)	**Test group**		**Control group**	
	No	**%**	**No**	**%**
Normal	94	72.9	67	72.0
TC ↑	2	1.6	1	1.1
LDL ↑	30	23.3	24	25.8
TGL ↑	9	7.0	7	7.5
HDL ↑	11	8.5	7	7.5
Total abnormal cases	35	27.1	26*	28.0
Total cases	129	100	93*	100
'p'	0.9019 Not significant			

Out of 129 women in the test group, 35 cases (27.1%) had abnormal lipid profile and out of 93 women in the control group 26 cases (28%) of them had elevated lipid profile values.

LDL was the most elevated than total cholesterol and triglycerides.

The 'p' value was 0.9019 which was not statistically significant.

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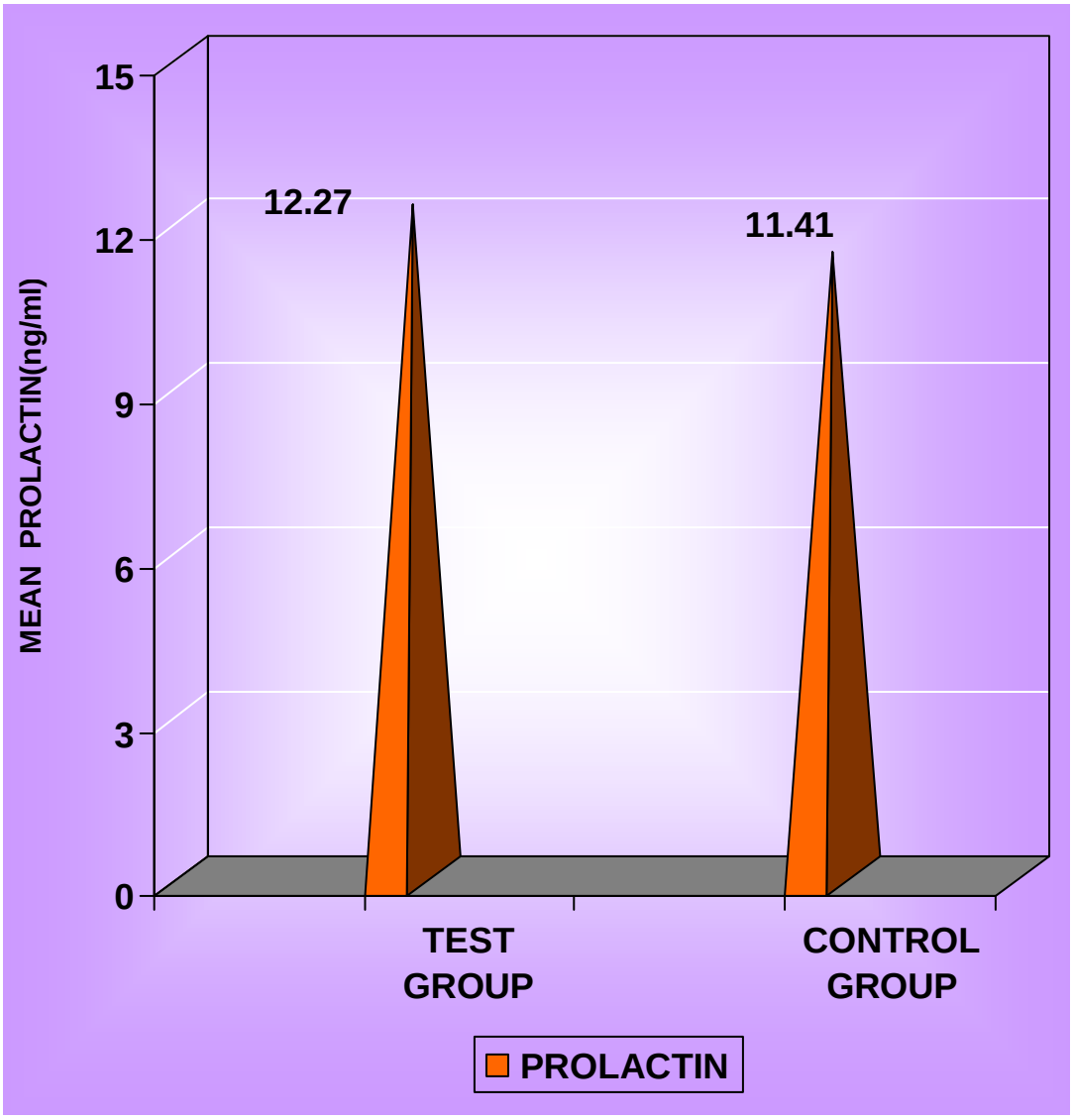
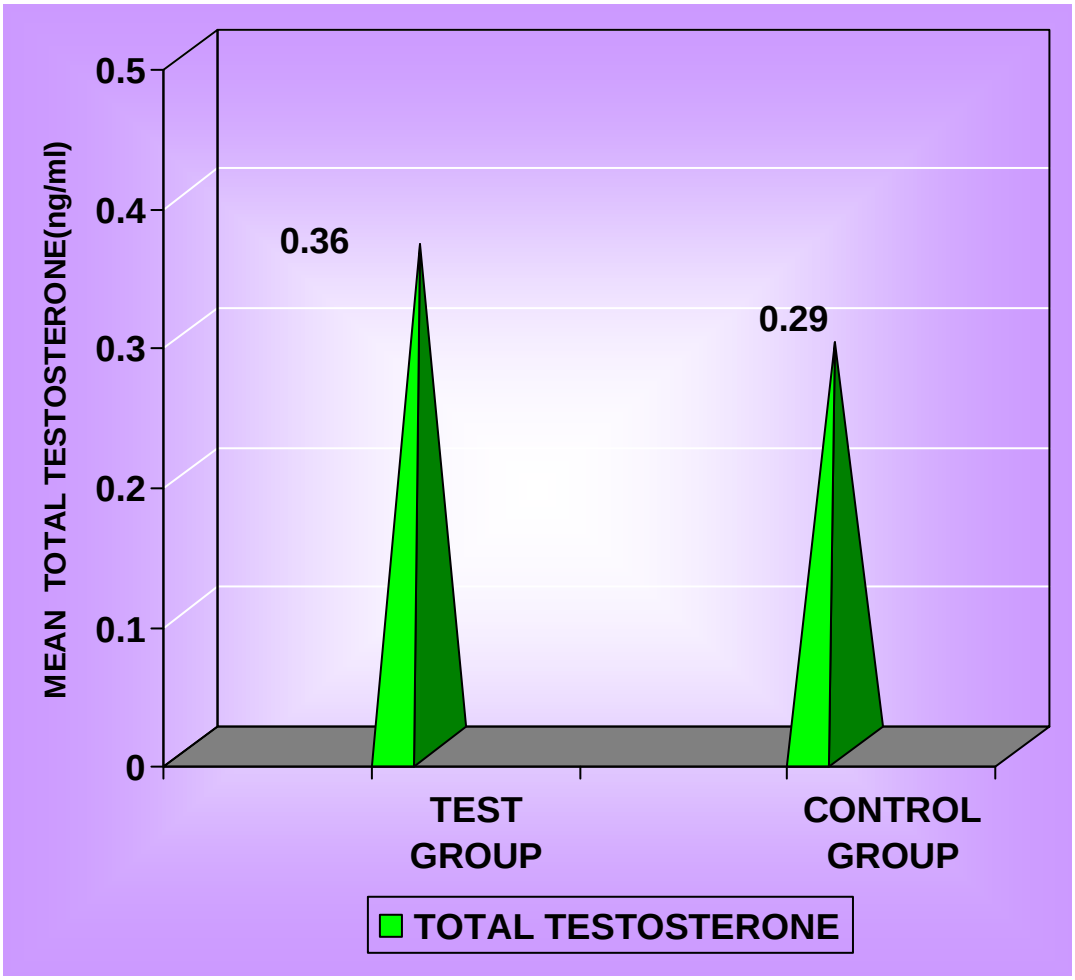
PROLACTIN



Group	**Prolactin (mg/ml)**					
	Mean	**SD**	**Normal**		**Abnormal**	
			No	**%**	**No**	**%**
Test group	12.17	7.5	117	90.7	12	9.3
Control	11.41	8.16	81	87.1	12	12.9
'p'	0.0869 Not significant					

The mean prolactin levels in the test group and control group is 12.17 and 11.41 respectively. The 'p' value is 0.0869 which is not significant.

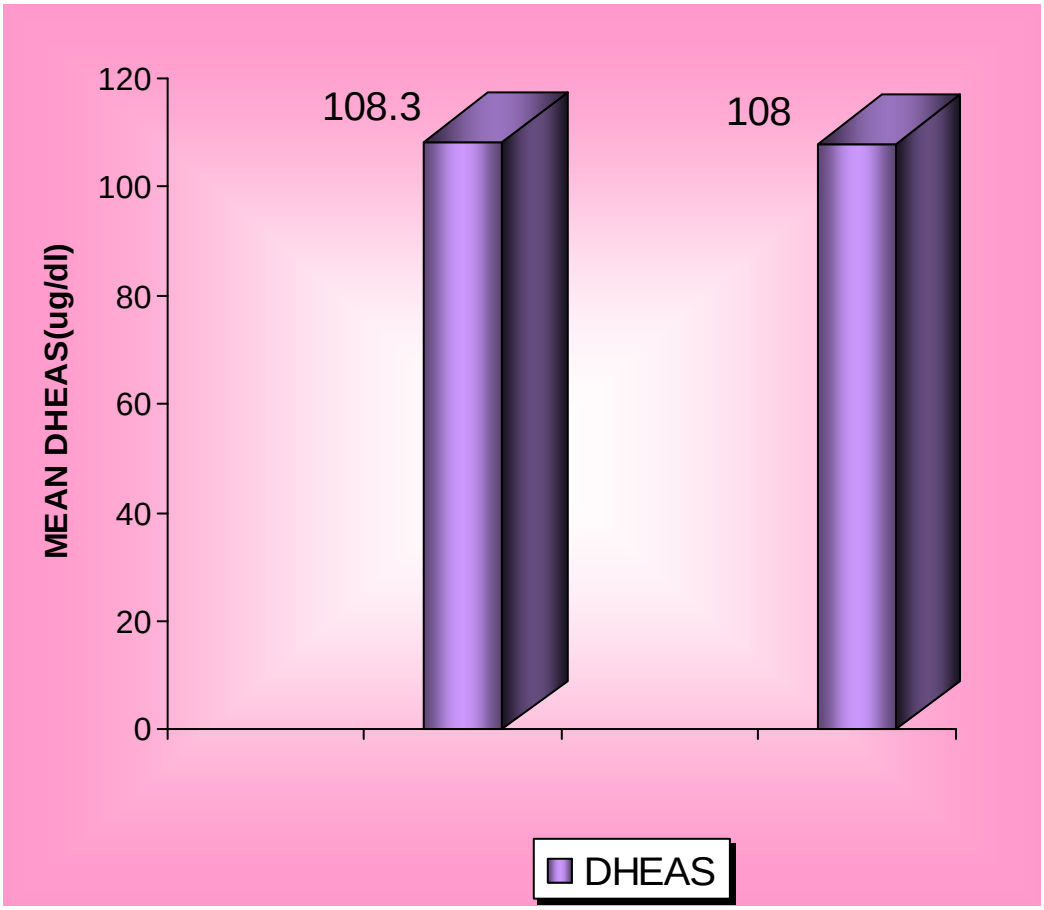
TOTAL TESTOSTERONE



Group	Total Testosterone (ng/dl)					
	Mean	SD	Normal		Abnormal	
			No	%	No	%
Test group	0.36	0.49	123	96.1	6	3.9
Control	0.29	0.18	92	98.9	1	1.1
'p'	0.719 Not significant					

The 'p' value comparing the mean and standard deviation between the test and control group was 0.719 for total testosterone levels. This is not statistically significant.

DHEAS



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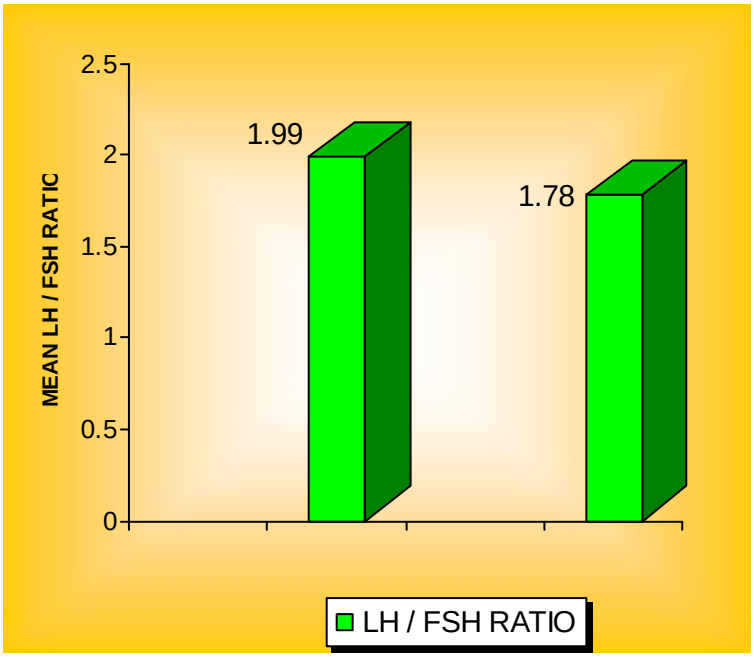
Group	DHEAS (ug/dl)					
	Mean	SD	Normal		Abnormal	
			No	%	No	%
Test group	108.3	78.1	126	97.7	2	2.3
Control	108.0	66.6	93	100	-	-
'p'	0.5493 Not significant					

Among the women with long cycles / scanty periods and those women with normal cycles there is no statistically significant difference in the DHEAS levels.

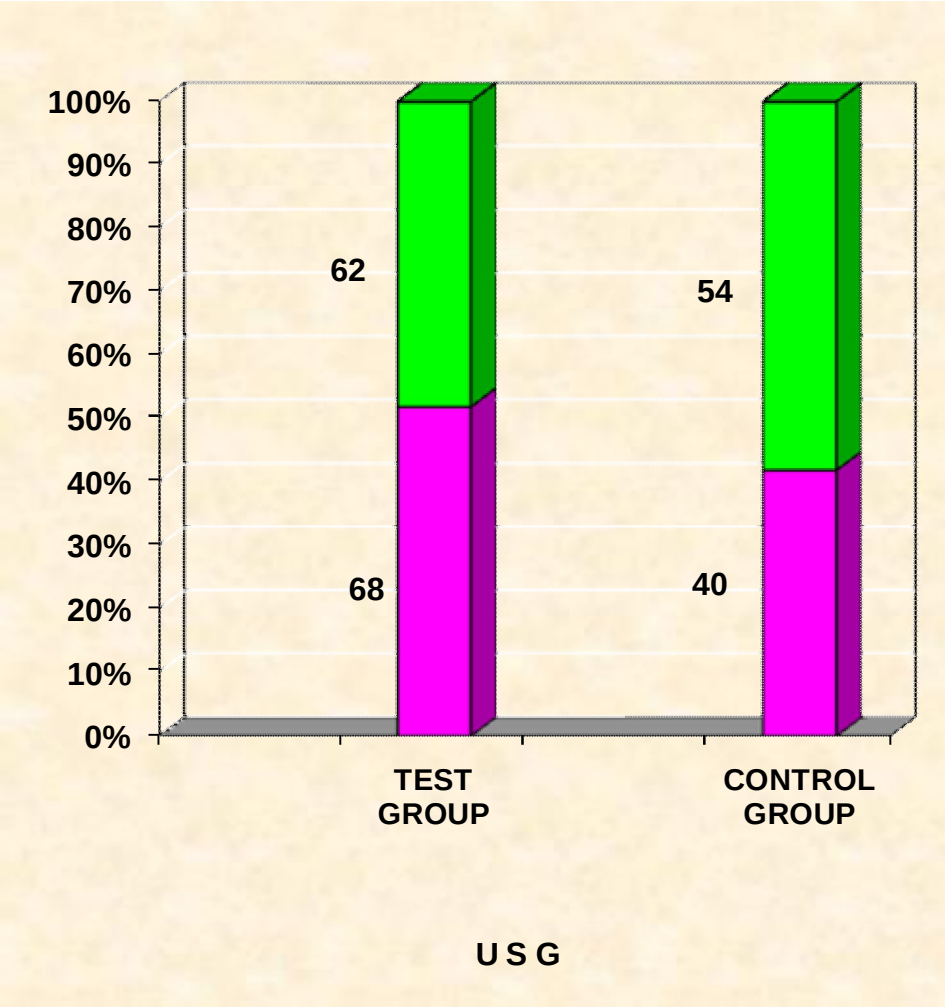
LH/FSH RATIO



Group	LH/FSH Ratio					
	Mean	SD	Normal		Abnormal	
			No	%	No	%
Test group	1.99	3.46	93	72.1	36	27.9
Control	1.78	1.84	67	72	26	28
'p'	0.9737 Not significant					

The ' p' value comparing the mean and standard deviation between the test and control group is 0.9737 which is greater than 0.05. Hence there is no statistically significant difference.

USG

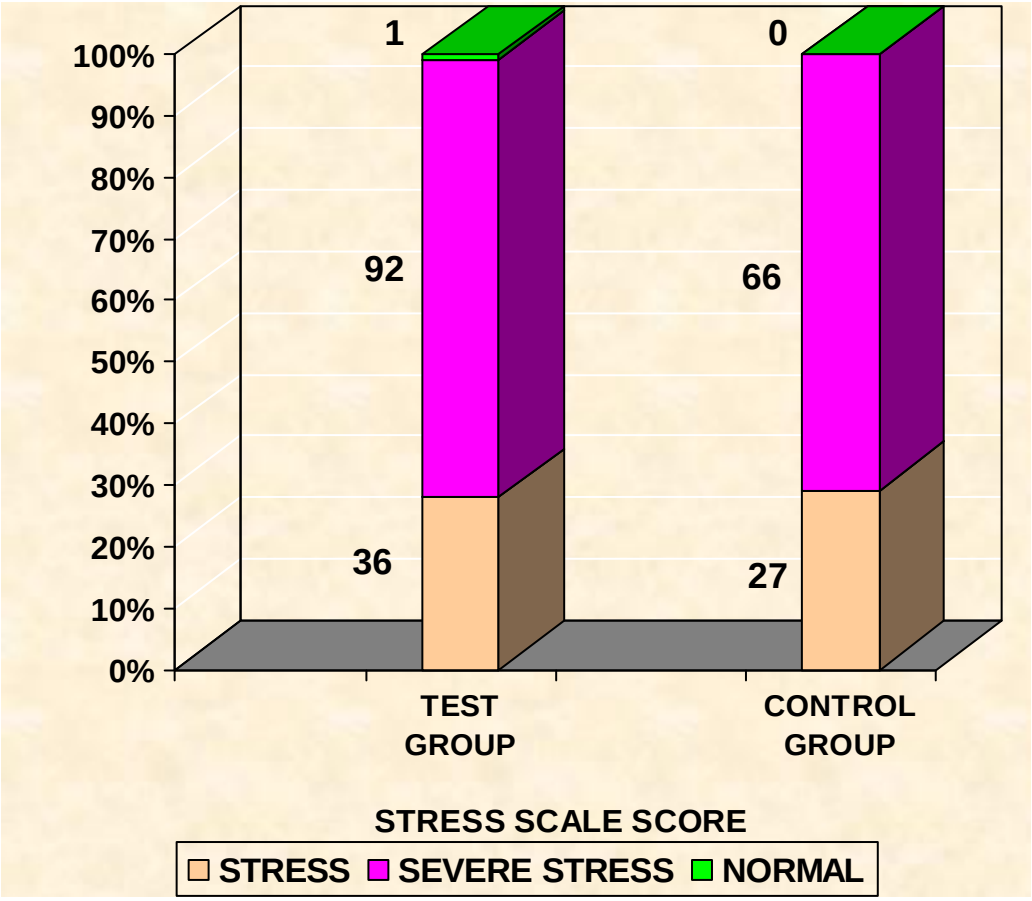

100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
62
68
54
40
TEST GROUP
CONTROL GROUP
U S G

USG	**Test group**		**Control group**	
	No	**%**	**No**	**%**
Bilateral PCOD	68	63	40	37
Normal	61	53.5	53	46.5
Total	129	100	93	100
‘p’	0.1967 Not significant			

In the long cycles / scanty periods, 68 had ultrasound features of bilateral PCOD. In those with regular cycles 40 had bilateral pcod on ultrasound.

The ‘ p’ value is 0.1967 and hence not significant

STRESS SCALE SCORE



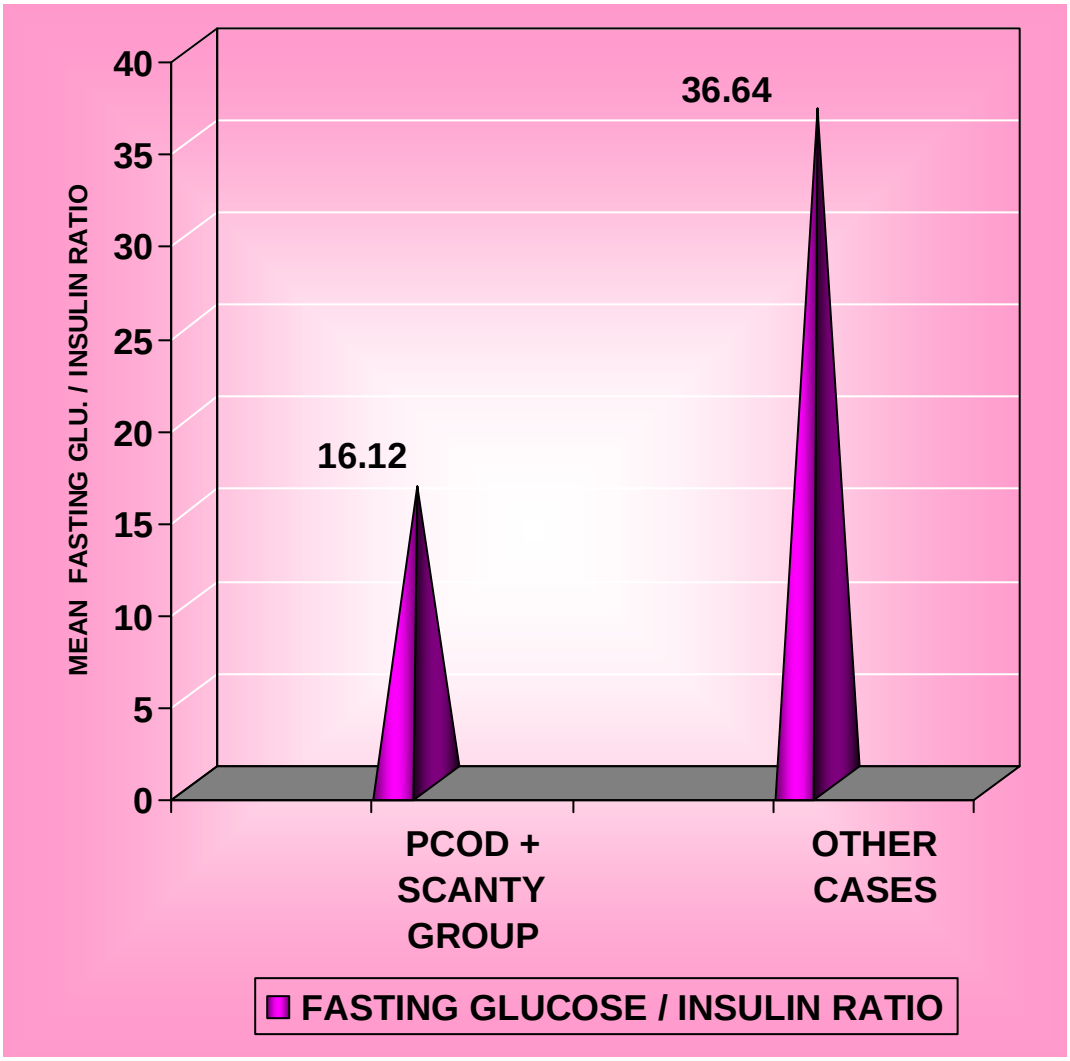
Stress scale score	**Test group**		**Control group**	
	No	**%**	**No**	**%**
Normal (<12)	1	0.8	-	-
Stress (12 – 20)	36	27.9	27	29
Severe stress (>20)	92	71.3	66	71
Score Mean SD	21.87 3.9		22.13 4.18	
'p'	0.7075 Not significant			

In women with scanty periods (129) there was 36 women with stress and 92 women with severe stress (71.8%).

In the control group there were 27 women (29%) with stress and 66 women (71%) with severe stress. The 'p' value is 0.7075.

The stress scale score also do not exhibit any statistically significant difference.

PCOS – FASTING GLUCOSE INSULIN RATIO

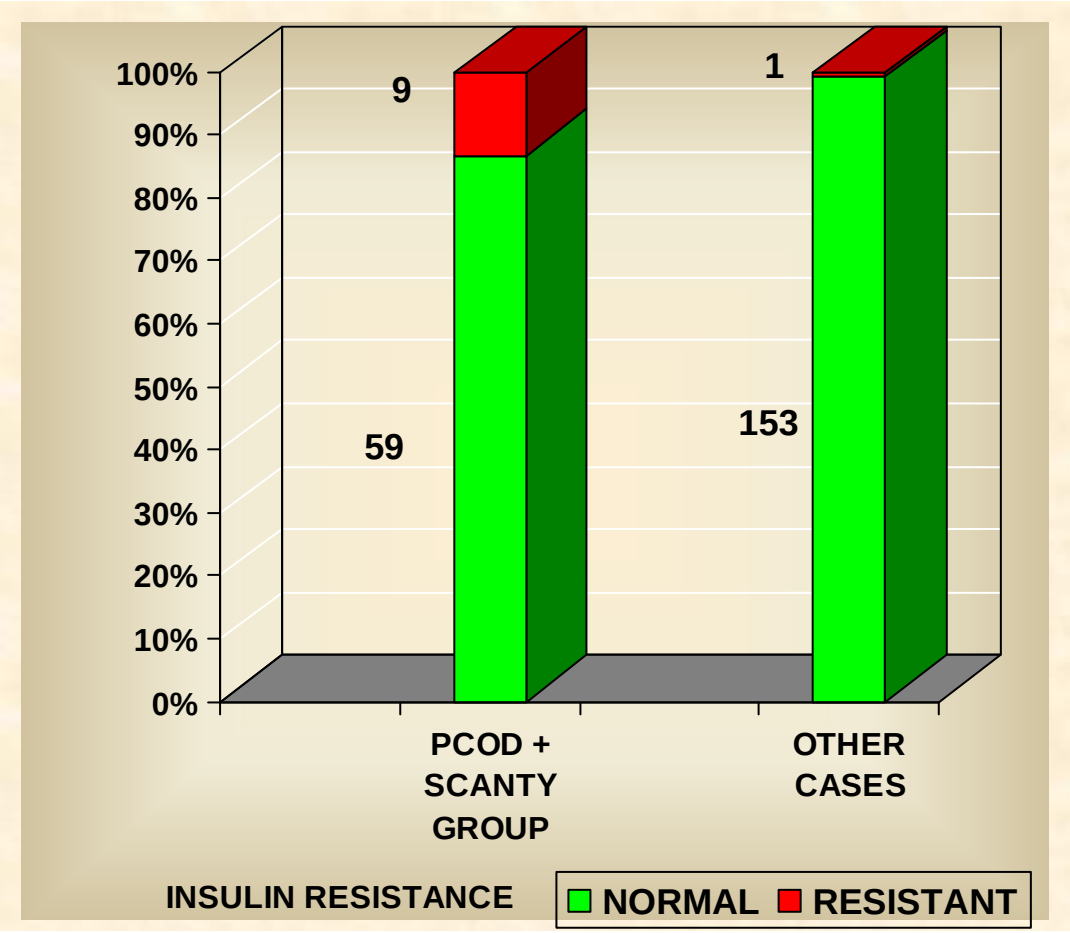


Fasting glucose / Insulin ratio

Fasting glucose insulin ratio	**PCOD + scanty cycles group**	**Other cases**
Mean	16.12	36.94
SD	7.9	34.57
'p'	**< 0.0001 Significant**	

The mean Fasting Glucose Insulin Ratio in the PCOS group is lower than that in the Non – PCOS group and the 'p' value is < 0.0001 which is statistically significant.

INSULIN RESISTANCE



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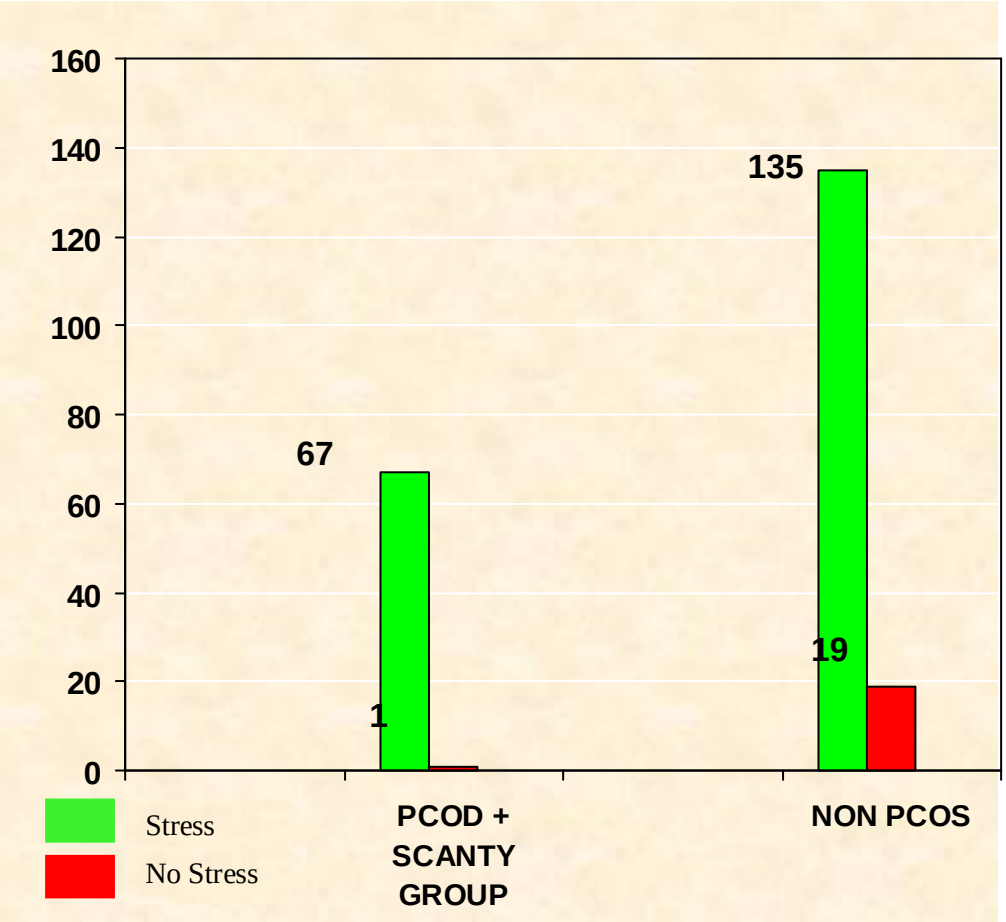
Group	**Insulin resistance**			
	Normal		**Resistant**	
	No	**%**	**No**	**%**
PCOD + scanty cycles group	59	86.8	9	13.2
Other cases	153	99.4	1	0.6
'p'	**0.0001 Significant**			

Out of 68 cases in the PCOS group , 9 are insulin resistant (i.e) 13.2%. I in the Non – PCOS group is resistant (i.e) 0.6 %. The 'p' value is 0.0001 which is statistically significant.

STRESS SCALE SCORE



Out of 68 PCOS cases, 67 of them had stress (i.e) 98.5% of them had stress score positive.

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RESULTS

In this study, out of 223 adults,

1 person was excluded because her blood sample was lysed.

8 of them had incomplete data with biochemical markers, still they were included in the study because with the available data insulin resistance could be calculated.

To increase the accuracy of the study, and due to availability of study cases, the subjects enrolled in the study group were 129 (i.e) with long cycles and scanty periods

In the control group there were 93 cases (i.e) with regular cycles.

Demographic variables, biochemical markers Stress scale Score, Clinical signs, Ultrasonogram features were compared between women with long cycles / scanty periods and with regular cycles.

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Demographic variables, Biochemical markers, Stress scale Score:

S.No	Variables	Long Cycles / Scanty Periods	Regular Cycles	P-Value
1	Age	20.4 +/-1.8	20.8+/-3.1	0.6947
2	Body Mass Index (Kg/M2)	21.92+/-2.22	21.87+/-2.11	0.7563
3	Glucose Fasting (mg/dl)	89.6+/-11.1	89.5+/-9.5	0.7227
4	2 Hour Glucose – After 75 gms Glucose (mg/dl)	102.8+/-18.3	101.5+/-25.3	0.2744
5	Fasting Insulin (ulU/ml)	4.92+/-3.56	4.72+/-3.49	0.4136
6	2 Hour Insulin- After 75 gms Glucose (mg / dl)	23.29+-29.84	18.55+/-25.57	0.2112
7	Fasting G: I Ratio	26.43+/-9.7	34.78+-6.4	0.0346
8	TSH	1.78+/-1.58	1.69+/-2.35	0.3296
9	Free T 4	0.91+/-0.19	0.19+/-0.12	0.2642
10	Prolactin	12.17+/-7.5	11.41+/-8.16	0.0869

S.No	Variables	Long Cycles / Scanty Periods	Regular Cycles	P-Value
11	Total Testetorone	0.36 + / - 0.49	0.29+/-0.18	0.719
12	DHEAS	108.3+/-78.1	108.0+/-66.6	0.5493
13	LH :FSH Ratio	1.99+/-3.46	1.78+/-1.84	0.9737
14	Stress Scale Score	21.87+/-3.9	22.13+/-4.18	0.7075

Lipid Profile:

Out of 129 women in the test group, 35 cases (27.1%) had abnormal lipid profile and out of 93 women in the control group 26 cases (28%) of them had elevated lipid profile values.

LDL was the most elevated than total cholesterol and triglycerides.

Clinical Signs:

Acne was the most common clinical sign noted when compared to Hirsutism and Acanthosis Nigricans. Total cases with clinical signs (35.2 % vs 28%).

Family History:

3 individuals in the test group and 1 individual in the control group had strong Family History of diabetes with either one or both parents being affected.

Ultrasonogram:

Bilateral polycystic ovaries were found in 68 cases in test group and 40 cases in the control group.

Insulin Resistance:

In the 129 young adults with long cycles and scanty periods, there were 9 individuals (7%) with a fasting Glucose Insulin ratio less than 4.5 and in the 93 with regular cycles there was one individual with Fasting Glucose Insulin Ratio less than 4.5 and she had both her parents on medications for Diabetes mellitus.

68 cases in the test group met the Rotterdam criteria for Polycystic ovarian syndrome with two out of three criteria positive (i.e) with oligo / anovulation (long cycles / scanty periods) and ultrasonogram criteria for PCOD.

PCOS group was compared with the non- PCOS group and results of insulin resistance was calculated

The mean Fasting Glucose : Insulin Ratio in PCOS group (16.12 + / - 7.9) was lower than that in non PCOS group (36.94 =/- 34.57). The 'P' value is <0.0001 which is Statistically significant.

TSH (5.8% VS 1.94) and Lipid Profile - Elevated LDL, TG(33.8% vs 26.6%) – were increased in the PCOS group.

Prolactin (2.94 % vs 5.6%) and LH:FSH ratio (13.2% vs 13.6%) – did not show any difference between both groups.

There were 2 cases in the PCOS group with elevated DHEAS, but none in the non – PCOS group.

Total Testosterone was normal in both the groups.

Regarding clinical signs, 38 out of 68 in PCOS group had one or more than one of the three clinical signs and again acne was the most common sign. Out of 6 total cases with Hirsutism only 1 met criteria of PCOS and the rest 5 had ultrasonogram features of PCOD. Out of 17 total cases with Acanthosis nigricans 8 were in the PCOS group.

Stress Scale Score:

Almost 98.5% with PCOS had Perceived Stress scale Score positive. 67 out of 68 had stress and 45 had scores in severe stress range.

Insulin Resistance:

Out of 68 cases with PCOS 9 individuals had a Fasting Glucose Insulin Ratio < 4.5 thus meeting the criteria of insulin resistance. In the Non – PCOS group 1 individual was insulin resistant and she had a strong family history with both parents having Type 2 Diabetes Mellitus.

Thus the prevalence of insulin resistance in polycycstic ovarian syndrome found by this study is 13.2 % and the 'P' value is 0.0001 which is statistically significant.

Thus this study shows that the prevalence of insulin resistance in young adults with abnormal menstrual pattern (i.e.) those with long cycles / scanty periods is 7% and the prevalence of insulin resistance in young adults with PCOS 13.2 %. Thus this is a cross sectional survey with nested case control study.

The Stress Scale Score showed a 98.5 % association in those who were insulin resistant.

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DISCUSSION

Oligo ovulation (long cycles / scanty periods) and Insulin resistance are markers which can predict future Type 2 Diabetes mellitus, cardiovascular disease, endometrial cancer, infertility, pregnancy complications in adults at a younger age.

Identifying these markers paves way for counselling, life style changes making individuals modify the natural course of the disease help in secondary prevention in a small subset of population.

Hyperinsulinemia and insulin resistance associated with long cycles / scanty periods along with ultrasound detection of polycystic ovaries are the underlying abnormalities in Polycystic Ovarian Syndrome.

Insulin resistance induces unfavourable changes in the lipid metabolism and also increase androgen production from theca cells.

This is a vicious cycle and interruption of this cycle at any point prevents or delays the complications.

The prevalence of Polycystic ovarian syndrome in lean individuals -20% (Robert Shaw Text book of Gynaecology).[77]

In the present study the mean Body Mass Index is 21.92 +/ - 2.22, (i.e.) lean individuals, the prevalence of PCOS - 30%.

Insulin Resistance was calculated using Fasting Glucose Insulin Ratio.

Leon Speroff - <4.5(1)

Legro et al - <4.5(24)

Kauffmann et al - <4.0(78)

Present study - <4.5

Jamal Golbahar 2012[26]	**Present Study**
Hyperinsulinemia was significantly and independently associated with PCOS	Hyperinsulinemia was significantly and independently associated with PCOS

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Rai et al[79]	**Present study**
Insulin resistance prevalence in PCOS was 76.9% using a Fasting Glucose Insulin Ratio <4.5	Insulin resistance prevalence in PCOS was 13.2% using a Fasting Glucose Insulin Ratio < 4.5
PCOS with insulin resistance had dyslipidemia	PCOS with insulin resistance had dyslipidemia

De Ugarte et al[80]	Insulin Resistance using HOMA-IR -64%
Ponte et al [81]	Insulin resistance using Fasting glucose Insulin Ratio - 56%
Present study	Insulin resistance using Fasting Glucose Insulin Ratio is 13.2%

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The previous studies showed a higher prevalence of insulin resistance. This may be due to the age group difference between the previous studies and the present study.

In this study the age group included were those only in the 18 – 24 years (i.e.) only young adults were included. This age group parameter was not found in the previous studies. This may explain the lower prevalence of insulin resistance when compared with the previous studies.

Dewailly et al – the Rotterdam criteria definition recognizes four PCO syndrome.

Hyperandrogenism + Polycystic ovaries + Oligoovulation – Full blown syndrome.

Hyperandrogenism + Oligoovulation - (National Institutes of Health definition).

Hyperandrogenism + Polycystic ovaries – Ovulatory PCOS.

Oligoovulation + Polycystic ovaries – have mild endocrine and metabolic features of PCOS.

The present study falls in the fourth category as in Dewailly et al study, as hyperandrogenemia was not evident in the present study[10].

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Sinha et al found a higher mean TSH level in PCOS patient. In the present study also mean TSH level is high.(5.8% vs 1.94%)[51].

The present study shows no correlation between LH : FSH ratio with PCOS, which was shown by Golbahar et al, that LH:FSH ratio is independently associated with PCOS.[26]

Gita et al, showed that there is a 15% increase in prolactin levels in PCOS.[2] The present study does not show any correlation between Prolactin levels and PCOS.

Angela Kerschner et al 2009, Fetil Steril 2009 – showed a significant high risk of mood disorders in patients with PCOS.The present study also showed a 71.8% association of psychological stress in those with long cycles/ scanty periods and a surprising 98.5% association with those having PCOS.

SUMMARY

This cross sectional survey with nested case control study was done in the Department of Obstetrics and Gynaecology, ESI Medical College and PGIMSR, in association with 11 health camp visits conducted at an export factory, MEPZ, Tambaram, Chennai done between August 2012 – August 2013.These subjects were ESI beneficiaries.

A total of 223 young adults in the age group of 18 – 24 were recruited for the study after informed consent and a questionnaire on the menstrual pattern of adults working in the export factory.

Among them 129 with long cycles / scanty periods were in the test group and 93 in the control group.

More number of subjects was enrolled in the study group to increase the accuracy of the study and due to availability of subjects and after the consultation with a Statistician.

All subjects had a detailed clinical examination, questionnaire for stress scale score, biochemical investigations, and ultra sonogram. Among them 68 met the criteria for PCOS fulfilling 2 of the three of

Rotterdam criteria. These subjects had oligo ovulation (i.e 0 long cycles / scanty periods) and ultrasound criteria of Bilateral PCOD.

As per the Aim of the study insulin resistance was calculated first for the 129 with long cycles and scanty periods and then for those with Polycystic ovarian syndrome.

There was a 7% prevalence of insulin resistance in individuals with long cycles / scanty periods (i.e.) abnormal menstrual pattern.

There was a 13.2 % prevalence of insulin resistance in those who met the criteria of PCOS.

There was a 71.8% association of stress with long cycles / scanty periods and 95.8% association of stress in those with PCOS.

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CONCLUSION

It is observed from the present study that insulin resistance manifests at an early age in women with polycystic ovarian syndrome. Oligoovulation (Long cycles / scanty periods) is a useful marker to identify subjects with insulin resistance.

Counselling regarding Life style modifications, maintaining BMI should be made available to all women who are insulin resistant.

Thus by early diagnosis at an asymptomatic stage , Sprogression of the disease can be halted.

BIBLIOGRAPHY

1. Speroff L. Clinical Gynaecologic Endocrinology and Infertility. Eight.

2. Gita Ganguly Mukherjee. Polycystic ovary syndrome -An Update. Jaypee (FOGSI);

3. Social and Preventive Medicine - Park.

4. A Dunaif AT. Current concepts in the polycystic ovary syndrome. Annu Rev Med. 2001; 52:401–19.

5. Pauli JM, Raja-Khan N, Wu X, Legro RS. Current perspectives of insulin resistance and polycystic ovary syndrome. Diabet Med J Br Diabet Assoc. 2011 Dec; 28(12):1445–54.

6. Treloar AE, Boynton RE, Behn BG, Brown BW. Variation of the human menstrual cycle through reproductive life. Int J Fertil. 1967 Mar; 12(1 Pt 2):77–126.

7. A E Treloar REB. Variation of the human menstrual cycle through reproductive life. Int J Fertil. 1968; 12(1 Pt 2):77–126.

8. Wood JW. Dynamics of Human Reproduction: Biology, Biometry, Demography. Transaction Publishers; 1994.

ABBYY PDF Transformer 2.0 Click here to buy www.ABBYY.com

9. Kase NG. Chronic an ovulation and the polycystic ovary syndrome. Altchek %27s Diagnosis and Management of Ovarian Disorders.

10. Dewailly D, Catteau-Jonard S, Reyss A-C, Leroy M, Pigny P. Oligoanovulation with Polycystic Ovaries But Not Overt Hyperandrogenism. J Clin Endocrinol Metab. 2006 Oct 1; 91(10):3922–7.

11. Literature Review Polycystic Ovarian Syndrome Health And Social Care Essay [Internet]. [cited 2013 Dec 1].

12. Unluturk U, Harmanci A, Kocaefe C, Yildiz BO. The Genetic Basis of the Polycystic Ovary Syndrome: A Literature Review Including Discussion of PPAR-γ.

13. Legro RS, RS L. Molecular progress in infertility: polycystic ovary syndrome. Fertil Steril. 2002 Sep;78(3):569–76.

14. Vrbíková J, Bendlová B, Hill M, Vanková M, Vondra K, Stárka L. Insulin Sensitivity and β-Cell Function in Women With Polycystic Ovary Syndrome. Diabetes Care. 2002 Jul 1;25(7):1217–22.

15. Begum F. Clinical and Hormonal Profile of Polycystic Ovary Syndrome. J South Asian Fed Obstet Gynecol. 2009 Aug 25;1(2):22–5.

16. Ramanand SJ, Ghongane BB, Ramanand JB, Patwardhan MH, Ghanghas RR, Jain SS. Clinical characteristics of polycystic ovary

syndrome in Indian women. Indian J Endocrinol Metab. 2013;17(1):138–45.

17. Chhabra S, Venkatraman S. Menstrual dysfunction in rural young women and the presence of polycystic ovarian syndrome. J Obstet Gynaecol J Inst Obstet Gynaecol. 2010 Jan;30(1):41–5.

18. Prevalence of polycystic ovary syndrome in young women from North India: A Community-based study Gill H, Tiwari P, Dabadghao P - Indian J Endocr Metab.

19. Huang A, Brennan K, Azziz R, Huang A BK. Prevalence of hyperandrogenemia in the polycystic ovary syndrome diagnosed by the National Institutes of Health 1990 criteria. Fertil Steril. 2010 Apr;93(6):1938–41.

20. Pearson S, Schmidt M, Patton G, Dwyer T, Blizzard L, Otahal P, et al. Depression and Insulin Resistance Cross-sectional associations in young adults. Diabetes Care. 2010 May 1;33(5):1128–33.

21. Carmina E, E C. Diagnosis of polycystic ovary syndrome: from NIH criteria to ESHRE-ASRM guidelines. Minerva Ginecol. 2004 Feb;56(1):1–6.

22. Teede H, Deeks A, Moran L. Polycystic ovary syndrome: a complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. BMC Med. 2010 Jun 30;8(1):41.

23. Prapas N, Karkanaki A, Prapas I, Kalogiannidis I, Katsikis I, Panidis D. Genetics of Polycystic Ovary Syndrome. Hippokratia. 2009;13(4):216–23.

24. Legro RS, Finegood D, Dunaif A. A fasting glucose to insulin ratio is a useful measure of insulin sensitivity in women with polycystic ovary syndrome. J Clin Endocrinol Metab. 1998 Aug;83(8):2694–8.

25. Vuguin P, Saenger P, Dimartino-Nardi J. Fasting glucose insulin ratio: a useful measure of insulin resistance in girls with premature adrenarche. J Clin Endocrinol Metab. 2001 Oct;86(10):4618–21.

26. Golbahar J, Al-Ayadhi, Das, Gumma. Sensitive and specific markers for insulin resistance, hyperandrogenemia, and inappropriate gonadotrophin secretion in women with polycystic ovary syndrome: a case-control study from Bahrain. Int J Womens Health. 2012 May;201.

27. Kauffman RP, Baker VM, Dimarino P, Gimpel T, Castracane VD. Polycystic ovarian syndrome and insulin resistance in white and Mexican American women: a comparison of two distinct populations. Am J Obstet Gynecol. 2002 Nov;187(5):1362–9.

28. Mather KJ, Kwan F, Corenblum B, Mather KJ KF. Hyperinsulinemia in polycystic ovary syndrome correlates with increased cardiovascular risk independent of obesity. Fertil Steril. 2000 Jan;73(1):150–6.

ABBYY PDF Transformer 2.0 Click here to buy www.ABBYY.com

29. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report-20110602_the_androgen_excess_and_pcos_society.pdf

30. A direct effect of hyperinsulinemia on serum sex ormone-binding globulin levels in obese women with pcos j clin endocrinol metab 1991; 72: 83-9 - Google Search.

31. ovultion induction: basic science and clinical advances Amsterdam: Elsevier science BV 1994; 103-14 - Google Search [Internet]. [cited 2013 Dec 6].

32. Insulin regulation of insulin like growth factor binding protein 1J clin endocrinol metab 1991;72(10 83-9 - Google Search.

33. Fulghesu AM, Angioni S, Portoghese E, Milano F, Batetta B, Paoletti AM, et al. Failure of the homeostatic model assessment calculation score for detecting metabolic deterioration in young patients with polycystic ovary syndrome. Fertil Steril. 2006 Aug;86(2):398–404.

34. Angioni S, Sanna S, Magnini R, Melis GB, Fulghesu AM. The quantitative insulin sensitivity check index is not able to detect early metabolic alterations in young patients with polycystic ovarian syndrome. Gynecol Endocrinol Off J Int Soc Gynecol Endocrinol. 2011 Jul;27(7):468–74.

35. Insulin stimulates androgen accumulation in incubations of ovarian stroma obtained from women with hyperandrogenism. J clin endocrinol metab 1986; 62: 904-10 - Google Search

36. G S Conway PMC. Hyperinsulinaemia in the polycystic ovary syndrome confirmed with a specific immunoradiometric assay for insulin. Clin Endocrinol (Oxf). 1993;38(2):219–22.

37. Diagnosis of polycystic ovary syndrome in adults [Internet]. [cited 2013 Dec 1].

38. McAuley KA, Williams SM, Mann JI, Walker RJ, Lewis-Barned NJ, Temple LA, et al. Diagnosing Insulin Resistance in the General Population. Diabetes Care. 2001 Mar 1;24(3):460–4.

39. Solomon CG, Hu FB, Dunaif A, Rich-Edwards JE, Stampfer MJ, Willett WC, et al. Menstrual cycle irregularity and risk for future cardiovascular disease. J Clin Endocrinol Metab. 2002 May;87(5):2013–7.

40. Sengupta. Gynaecology For Postgraduate And Practitioners. Elsevier India; 2007.

41. Diagnostic Criteria in Polycystic Ovary Syndrome: Insulin Resistance [Internet]. [cited 2013 Dec 6].

42. Michelmore KF, Balen AH, Dunger DB, Vessey MP. Polycystic ovaries and associated clinical and biochemical features in young women. Clin Endocrinol (Oxf). 1999 Dec;51(6):779–86.

ABBYY PDF Transformer 2.0 Click here to buy www.ABBYY.com

43. Adashi EY, Thorner MO, USA SS. The Somatotrophic Axis and the Reproductive Process in Health and Disease. Springer; 1995.

44. Low dose combination of flutamids, metformin and an ocp in obese young women with pcos hum repro 2003 - Google Search

45. Ja E. The polycystic ovary syndrome presenting as resistant acne successfully treated with cyproterone acetate. Med J Aust. 1991 Nov;155(10):677–80.

46. low dose combination of flutamids, metformin and an ocp in obese young women with pcos hum repro 2003 - Google Search

47. Puri N. A study of pathogenesis of acanthosis nigricans and its clinical implications. indian j Dermatol. 2011;56(6):678–83.

48. Dunaif A, Hoffman AR, Scully RE, Flier JS, Longcope C, Levy LJ, et al. Clinical, biochemical, and ovarian morphologic features in women with acanthosis nigricans and masculinization. Obstet Gynecol. 1985 Oct;66(4):545–52.

49. Acanthosis nigricans - Wikipedia, the free encyclopedia [Internet]. [cited 2013 Dec 6].

50. Sheehan MT. Polycystic Ovarian Syndrome: Diagnosis and Management. Clin Med Res. 2004 Feb;2(1):13–27.

51. Sinha U, Sinharay K, Saha S, Longkumer TA, Baul SN, Pal SK. Thyroid disorders in polycystic ovarian syndrome subjects: A

ABBYY PDF Transformer 2.0 Click here to buy www.ABBYY.com

tertiary hospital based cross-sectional study from Eastern India. Indian J Endocrinol Metab. 2013;17(2):304–9.

52. Hyperprolactinaemia and Prolactinoma | Doctor [Internet]. Patient.co.uk. [cited 2013 Dec 1].

53. Evidence for association between pcos and premature carotid arterioscerosis in middle aged women arterioscler thromb vas biol 2000 - Google Search [Internet]. [cited 2013 Dec 6].

54. Image: Exclusion of Androgen Excess Disorders other than PCOS [Internet].

55. Kyei-Mensah A, Zaidi J, Campbell S. Ultrasound diagnosis of polycystic ovary syndrome. Baillières Clin Endocrinol Metab. 1996 Apr;10(2):249–62.

56. Swanson M, Sauerbrei EE, Cooperberg PL. Medical implications of ultrasonically detected polycystic ovaries. J Clin Ultrasound JCU. 1981 Jun;9(5):219–22.

57. Journal of medicine and life | Insulin resistance and fertility in polycystic ovary syndrome [Internet]. [cited 2013 Dec 6].

58. Dunaif A. Polycystic Ovary Syndrome: Current Controversies, from the Ovary to the Pancreas. Springer; 2008.

59. Legro RS. Insulin resistance in women's health: why it matters and how to identify it. Curr Opin Obstet Gynecol. 2009 Aug;21(4):301–5.

ABBYY PDF Transformer 2.0 Click here to buy www.ABBYY.com

60. Prevalence of impaired glucose tolerance and diabetes in women with pcos Diabetes care 1999 - Google Search [Internet]. [cited 2013 Dec 6].

61. Prevalence of impaired glucose tolerance and diabetes in women with pcos Diabetes care 1999 - Google Search [Internet]. [cited 2013 Dec 6].

62. Legro RS, Kunselman AR, Dodson WC, Dunaif A. Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective, controlled study in 254 affected women. J Clin Endocrinol Metab. 1999 Jan;84(1):165–9.

63. Wild RA, Painter PC, Coulson PB, Carruth KB, Ranney GB. Lipoprotein Lipid Concentrations and Cardiovascular Risk in Women with Polycystic Ovary Syndrome. J Clin Endocrinol Metab. 1985 Nov 1;61(5):946–51.

64. Talbott EO, Guzick DS, Sutton-Tyrrell K, McHugh-Pemu KP, Zborowski JV, Remsberg KE, et al. Evidence for association between polycystic ovary syndrome and premature carotid atherosclerosis in middle-aged women. Arterioscler Thromb Vasc Biol. 2000 Nov;20(11):2414–21.

65. Cardiovascular Risk in PCOS [Internet]. [cited 2013 Dec 6].

ABBYY PDF Transformer 2.0 Click here to buy www.ABBYY.com

66. Cho LW, Randeva HS, Atkin SL. Cardiometabolic aspects of polycystic ovarian syndrome. Vasc Health Risk Manag. 2007 Feb;3(1):55–63.

67. Wild S, Pierpoint T, McKeigue P, Jacobs H. Cardiovascular disease in women with polycystic ovary syndrome at long-term follow-up: a retrospective cohort study. Clin Endocrinol (Oxf). 2000 May;52(5):595–600.

68. Rai R, Backos M, Rushworth F, Regan L. Polycystic ovaries and recurrent miscarriage--a reappraisal. Hum Reprod Oxf Engl. 2000 Mar;15(3):612–5.

69. Radon PA, McMahon MJ, Meyer WR. Impaired glucose tolerance in pregnant women with polycystic ovary syndrome. Obstet Gynecol. 1999 Aug;94(2):194–7.

70. Polycystic Ovary Syndrome□: A Major Cause Of Female Infertility [Internet]. [cited 2013 Nov 30].

71. Gill H, Tiwari P, Dabadghao P. Prevalence of polycystic ovary syndrome in young women from North India: A Community-based study. Indian J Endocrinol Metab. 2012 Dec;16(Suppl 2):S389–392.

72. Microsoft Word - J519 - habt04i3p126.pdf [Internet]. [cited 2013 Dec 6].

ABBYY PDF Transformer 2.0 Click here to buy www.ABBYY.com

73. Rodriguez D. PCOS and Endometrial Cancer Risk - Uterine Cancer Center - Everyday Health [Internet]. EverydayHealth.com. [cited 2013 Nov 30].

74. Polycystic Ovarian Syndrome: What Are the Risks To Your Health? [Internet]. [cited 2013 Dec 1].

75. Unfer V, Zacchè M, Serafini A, Redaelli A, Papaleo E, Unfer V ZM. Treatment of hyperandrogenism and hyperinsulinemia in PCOS patients with essential amino acids. A pilot clinical study. Minerva Ginecol. 2008 Oct;60(5):363–8.

76. Macut D, Pfeifer M, Yildiz BO, Diamanti-Kandarakis E. Polycystic Ovary Syndrome: Novel Insights Into Causes and Therapy. Karger Medical and Scientific Publishers; 2013.

77. Shaw R. Gynaecology. Fourth.

78. Kauffman RP, Baker VM, Dimarino P, Gimpel T, Castracane VD. Polycystic ovarian syndrome and insulin resistance in white and Mexican American women: a comparison of two distinct populations. Am J Obstet Gynecol. 2002 Nov;187(5):1362–9.

79. Rai L, Kalra A, Nair S. Association of obesity and insulin resistance with dyslipidemia in Indian women with polycystic ovarian syndrome. Indian J Med Sci. 2006;60(11):447.

ABBYY PDF Transformer 2.0 Click here to buy www.ABBYY.com

80. DeUgarte CM, Bartolucci AA, Azziz R. Prevalence of insulin resistance in the polycystic ovary syndrome using the homeostasis model assessment. Fertil Steril. 2005 May;83(5):1454–60.

81. Pontes AG, Rehme MFB, Martins AMV de C, Micussi MTABC, Maranhão TM de O, Pimenta W de P, et al. [Insulin resistance in women with polycystic ovary syndrome: relationship with anthropometric and biochemical variables]. Rev Bras Ginecol E Obstetrícia Rev Fed Bras Soc Ginecol E Obstetrícia. 2012 Feb;34(2):74–9.

PROFORMA

NAME :

AGE :

OCCUPATION :

HEIGHT : WEIGHT : BMI :

AGE AT MENARCHE :

CYCLE PATTERN :

LAST MENSTRUAL PERIOD :

GENERAL EXAMINATION :

BREAST :

THYROID :

ACNE :

HIRSUTISM :

ACANTHOSIS NIGRICANS :

FAMILY HISTORY :

PERCEIVED STRESS SCALE SCORE (QUESTIONNAIRE)

The questions in this scale ask you about your feelings and thoughts during the last month.

0 = Never
1 = Almost Never
2 = Sometimes
3 = Fairly Often
4 = Very Often

1) In the last mont, how often have you been upset because of something that happened unexpectedly?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

2) In the last month, how often have you felt that you were unable to control the important things in your life.

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

3) In the last month, how often have you felt nervous and “stressed”?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

4) In the last month, how often have you felt confident about your ability to handle your personal problems?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

5) In the last month, how often have you felt that things were going your way?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

6) In the last month, how often have you found that you could not cope with all the things that you had to do?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

7) In the last month, how often have you been able to control irritations in your life?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

8) In the last month, how often have you felt that you were on top of things?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

9) In the last month, how often have you been angered because of things that were outside of your control?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

10) In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

0=Never 1=Almost Never 2=Sometimes 3=Fairly Often 4=Very Often

Perceived Stress Scale Scoring

PSS – 10 scores obtained by receiving the scores on the four positive items e.g.,

0=4, 1=3, 2=2, etc., and then summing across all 10 items. Items 4, 5, 7 and 8 are the positively stated items.

BIOCHEMICAL PARAMETERS:

FASTING GLUCOSE

2HOUR GLUCOSE LEVEL – AFTER 75 GRAMS ORAL GLUCOSE

FASTING INSULIN

2 HOUR INSULIN LEVEL – AFTER 75 GRAMS ORAL GLUCOSE

SERUM PROLACTIN

SERUM TOTAL TESTOSTERONE

SERUM DHEAS

THYROID FUNCTION TESTS

SERUM LH

SERUM FSH

LH:FSH RATIO

LIPID PROFILE

ULTRASONOGRAPHY REPORT – PELVIS

UTERUS : Uterus appear verted and normal in size

Endometrium appear
Endometrial thickness is mm.
Myometrium appear
Focal lesion.

OVARIES	:	Right	Left
	Measures	mm.	
		Cc	
	No of follicles measuring		
	Between 2-mm		
	Dominant follicle size		
	Arrangement of follicles		
	Central stroma echogenecity		
	Ovarian outline		
	Hypoechoic ovaries		

POD free fluid

Impression: Radiologist

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ABBREVIATIONS

1.	PCOS	-	Polycystic Ovarian Syndrome
2.	WHO	-	World Health Organization
3.	(i.e)	-	That is
4.	LH	-	Luteinizing Hormone
5.	TSH	-	Thyroid Stimulating Hormone
6.	FSH	-	Follicular Stimulating Hormone
7.	BMI	-	Body Mass Index
8.	HDL	-	High Density Lipoprotein
9.	LDL	-	Low Density Lipoprotein
10.	TG	-	Triglycerides
11.	PPBS	-	Post Prandial Blood Sugars
12.	DHEAS	-	Dehydro epiandrosterone

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மருத்துவ மேற்படிப்பு ஆய்வால் ஏற்பட போகும் மேம்பாட்டை உணர்ந்து பொது நலன் கருதி என் சுயநினைவுடன் முழுமனதாக இந்த ஆய்வில் பங்கு பெற சம்மதிக்கிறேன்.

இதற்காக எனக்கு இரத்த பரிசோதனையும், கர்ப்பப்பை மற்றும் சூலகப்பகுதி ஸ்கேன் செய்யப்படும் என்பதை மருத்துவர் மூலம் அறிந்து கொண்டேன்.

இந்த பரிசோதனையின் அறிக்கைகளை நான் பெற்றுக்கொள்ளவும், ஏதேனும் குறைபாடுகள் இருந்தால் சிகிச்சை பெற்று கொள்ளவும் ஏற்பாடு செய்யப்படும் என்பதை மருத்துவர் தெரிவித்துள்ளார்.

இப்படிக்கு

CERTIFICATE OF APPROVAL

To

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ESI-PGIMSR, K.K.Nagar,
Chennai 600 078.

Dear Dr. R. Rajeswari,

The Institutional Ethics committee of ESI-PGIMSR, reviewed and discussed your application for approval of the proposal entitled **"A study on the prevalence of Insulin Resistance in Young Adults with Abnormal Menstrual Pattern"** No.5/03102012.

The following members of Ethics Committee were present in the meeting held on 03.10.2012 conducted at ESI-PGIMSR, Chennai 600 078.

1.	Dr. Saradha Suresh	-	Chairperson
2.	Dr. Kamalini Sridharan Prof. & HOD, Dept. Of Anesthesia, ESI-PGIMSR	-	Member Secretary
3.	Dr. Premila	-	EC Member
4.	Dr.N. Krishnan	-	EC Member
5.	Dr.K.S. Vijaya Raghavan	-	EC Member
6.	Dr.T.S. Swaminathan	-	EC Member
7.	Dr.C.V. Aravindan	-	EC Member
8.	Dr.S. Dhanalakshmi	-	EC Member
9.	Dr.B.S. Ayyappan	-	EC Member
10.	Dr.A. Sundaram	-	EC Member
11.	Dr.O.L. Naganath Babu	-	EC Member
12.	Sister Lalitha Teresa	-	EC Member

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

Prof. KAMALINI SRIDHARAN
M.D., D.A.,
DEPARTMENT OF ANAESTHESIA
ESI-PGIMSR,
ESIC HOSPITAL,
K.K. NAGAR, CHENNAI - 78.

Place : Chennai
Date : 03.10.2012

S.No	SID No	Age	BMI	Menstrual Pattern	Clinical Signs (acne,hirsutism, acanthosis nigricans)	USG	FASTING GLUCOSE (mg/dl)	PP GLUCOSE(mg /dl)	FASTING INSULIN (uIU/ml)	PP INSULIN (uIU/ml)	Fasting GLU : INS Ratio	TSH (mIU/L)	Free T 4 (ng/dl)	Lipid Profile (mg/dl)	Prolcatin (ng/ml)	Total Testosterone (ng/ml)	DHEAS (ug/dl)	LH /FSH Ratio	Stress Scale Score
1	59797	23	22.06	long,scanty	A +	B/L PCOD	95.4	108	5.42	21.62	17.6	0.9	0.91	Normal	10.5	0.18	105.7	0.8	17
2	59799	20	21.34	regular	N	NORMAL	91.7	95.1	2.05	11.55	44.7	2.03	0.95	Normal	4.54	0.43	169.4	3.6	27
3	59800	23	22.68	long,scanty	N	NORMAL	115	130	2.5	30.58	46	1.45	0.91	Normal	22.07	0.34	112.3	0.9	26
4	59801	21	21.09	long,scanty	N	NORMAL	108	103	2.15	32.2	50	1.5	0.99	Normal	12.14	0.33	118.3	4.84	17
5	59802	19	21.46	long,scanty	A+	B/L PCOD	97.8	121.6	2.75	42.08	35.6	1.54	0.93	Normal	11.24	0.34	67.4	5.89	27
6	59803	19	20.69	long,scanty	A+	B/L PCOD	93.2	98	4.95	53.36	18.8	1.3	1.14	Normal	12.99	0.69	34.1	0.92	25
7	59804	19	21.09	long,scanty	A+	NORMAL	79	92	2.23	6.45	35.4	0.96	0.77	↑/HDL ↓	4.47	0.13	70.3	0.89	26
8	59805	21	19.56	regular	N	NORMAL	88.9	101.7	0.58	5.91	151	0.8	0.92	Normal	5.15	0.17	86.1	1.49	25
9	59806	18	19.17	long,scanty	N	B/L PCOD	77.9	100.1	3	5.35	25.9	3.69	0.9	Normal	8.93	0.12	103.8	0.18	21
10	59807	19	22.23	long,scanty	N	NORMAL	96.1	102.4	2.43	16.1	39.5	1.47	0.88	Normal	18.01	0.39	87.9	3.64	24
11	59808	22	24.56	regular	N	B/L PCOD	96.2	163.8	4.27	214.1	22.5	2.38	0.94	Normal	26.64	0.53	377.3	4.03	21
12	59809	19	23.28	long,scanty	N	B/L PCOD	102	140	3.73	46.96	27.3	1.21	0.84	Normal	9.12	0.56	2213	3.28	18
13	59810	24	23.12	long,scanty	N	NORMAL	96.2	86.1	2.76	7.4	34.8	1.17	1.01	TGL ↑	13.15	0.22	80	0.88	17
14	59811	20	24.56	long,scanty	N	NORMAL	91.7	93.7	3.5	16.4	26.2	1.15	0.75	Normal	15.3	0.33	119.1	0.79	21
15	59812	24	19.74	regular	N	NORMAL	94	72.4	0.5	6.74	188	7.87↑	0.7	LDL ↑	10.8	0.34	290.3	0.52	22
16	59813	20	26.14	long,scanty	A+,MILD H+,AN+	B/L PCOD	109	110	5.54	7.84	19.6	3.06	0.89	Normal	17.8	0.77	148	4.03	13
17	59814	20	23.94	long,scanty	N	B/L PCOD	94	110	3.56		26.4	2.76	0.87	Normal	49.2 ↑	0.46	142.5	3.78	17
18	59815	22	25.87	`	N	NORMAL	103	111	4.07	16.49	25.3	1.67	0.82	Normal	42.6↑	0.44	78.1	0.69	16
19	59816	18	23.61	regular	N	NORMAL	99.6	89.4	1.92	4.12	51.8	1.35	0.9	Normal	18.6	0.22	75.3	0.63	22
20	59817	18	25.23	regular	A+,MILD H+	B/L PCOD	86.8	64.6	12.03	51.69	7.21	1.94	0.87	TGL ↑/ LDL ↑	9.71	0.43	185.3	0.71	17
21	59818	20	24.24	regular	N	NORMAL	89.3	91.1	2.47	14.9	36.1	2.37	1.04	Normal	15.7	0.22	71	2.12	22
22	59819	18	27.41	long,scanty	N	B/L PCOD	95.2	119.2	6.93	79.09	13.7	2.98	0.97	Normal	8.97	2.53	153.9	2.42	16
23	59820	20	27.77	long,scanty	A+	B/L PCOD	76.7	88.3	17.27	18.09	4.44	6.08 ↑	0.91	LDL ↑	9.53	0.49	114.9	0.3	22
24	59821	21	24.52	long,scanty	N	NORMAL	95	98	2.9	32.4	32.7	1.32	1.01	Normal	15.5	0.46	78.6	6.25	29
25	59822	23	26.3	long,scanty	N	B/L PCOD	113	92.9	6.73	57.6	16.7	1.85	0.75	LDL ↑	19.53	0.27	50.5	1.41	22
26	59823	20	21.79	regular	N	NORMAL	91.3	84.7	1.19	2.76	76.7	1.6	0.96	Normal	16.53	0.44	135.7	5.08	21
27	59824	20	20.61	long,scanty	N	NORMAL	100.3	92	1.86	3.61	53.9	1.06	0.89	Normal	23.1	0.28	70.8	2.26	23
28	59825	21	20.66	regular	A+	NORMAL	89	58.4	3.11	5.22	28.6	0.98	0.84	Normal	8.6	0.67	187.4	2.01	21
29	59826	19	24.67	regular	A+, AN+	B/L PCOD	90.7	92.5	6.74	5.29	13.4	1.87	1.2	Normal	5.98	0.43	138.6	2.01	20
30	59827	23	23.28	long,scanty	N	B/L PCOD	96.9	76.7	4.69	2.01	20.6	3.31	0.92	Normal	15.35	0.38	165.9	0.31	19
31	59828	19	21.97	regular	N	B/L PCOD	92.9	109.4	3.93	35.15	23.6	1.33	0.92	Normal	8.12	0.62	145.8	0.98	27
32	59829	19	21.22	regular	N	NORMAL	113.2	115.8	1.1	1.58	102	1.69	1.11	Normal	6.37	0.22	39.8	2.08	19
33	59830	20	22.23	regular	N	NORMAL	93.8	89.3	0.3	3.31	312	1.25	0.85	LDL ↑	22.3	0.44	111.6	3.25	24
34	59831	18	24.67	regular	A+	B/L PCOD	100.7	79.5	5.97	8.11	16.8	0.53	0.79	Normal	3.33	0.15	41.4	0.58	21

35	59832	22	21.45	long,scanty	N	NORMAL	89	99.4	0.79	3.88	119.6	4.09	0.88	↑/TGL ↑/ LDL	30.3	0.84	275.9	2.87	27
36	59833	19	21.56	long,scanty	N	NORMAL	94.1	83.4	1	1.22	94.1	0.8	0.88	Normal	12.9	0.53	253.4	1.84	19
37	59834	21	20.08	regular	N	NORMAL	93.4	72.1	0.75	0.77	124	1.41	0.98	Normal	8.03	0.58	132.7	0.97	21
38	59835	19	21.64	long,scanty	N	B/L PCOD	98.8	97.7	2.82	5.65	35	2.77	0.86	Normal	8.09	0.25	65.2	0.78	23
39	59836	24	23.45	long,scanty	N	B/L PCOD	68	89.8	16.96	4.76	4.01	9.22 ↑	0.81	↑/TGL ↑/ LDL	19.1	0.43	430 ↑	0.01	22
40	59837	20	24.14	long,scanty	A+,AN+	B/L PCOD	88	84.8	6.95	5.43	12.66	3.27	0.86	TGL ↑/ LDL ↑	12.27	0.04	134.8	0.83	21
41	59838	19	24.24	long,scanty	N	B/L PCOD	94.5	84.7	10.26	8.02	9.02	1.83	0.81	TGL ↑	7.78	0.37	62.5	3.04	21
42	59839	20	22.06	regular	N	NORMAL	90	88.6	1.37	12.83	65.6	1.68	1.05	LDL ↑	4.55	0.38	89.5	2.19	22
43	59840	19	19.91	long,scanty	N	NORMAL	87.9	67.9	0.66	0.84	133	0.8	0.86	Normal	12.3	0.44	81.8	2.86	25
44	61719	20	21.87	regular	N	NORMAL	93	86.4	2.26	2.44	41.1	1.66	0.89	Normal	7.49	0.22	64.5	1.27	21
45	61721	21	23.67	long,scanty	N	NORMAL	87	85.1	5.17	4.21	16.8	1.3	0.72	Normal	15.5	0.17	48.1	1.16	24
46	61724	23	20.96	long,scanty	N	NORMAL	95.2	89.8	1.91	6.85	49.8	2.12	0.87	Normal	8.06	0.27	72.6	0.76	19
47	61725	23	22.51	long,scanty	N	NORMAL	87.3	145.7	2.75	1.05	31.7	1.08	1	LDL ↑	9.6	0.35	54.5	2	19
48	61727	19	22.15	regular	N	NORMAL	100.9	90.3	3.21	1.99	31.4	2.76	0.72	Normal	5.79	0.29	78.9	0.711	18
49	61730	23	24.45	regular	A+	B/L PCOD	86.2	71.4	4.11	5.08	20.9	1.29	1.13	Normal	8.6	0.77	328.1	3.22	14
50	61731	22	21.5	long,scanty	N	NORMAL	89.4	82.8	2.73	9.06	32.7	0.38	0.78	normal	16.45	0.61	109.2	5.29 ↑	19
51	61732	19	21.05	long,scanty	N	NORMAL	90.3	102.9	2.61	9.58	34.5	0.56	0.99	normal	5.85	0.26	54.4	3.73	16
52	61733	20	22.21	regular	N	NORMAL	84.7	78.4	1.94	3.39	43.6	0.66	0.92	normal	2.51	0.22	63.7	1.42	20
53	61734	22	22.94	long,scanty	A+	B/L PCOD	74.9	84.8	16.69	6.23	4.48	0.65	0.86	LDL ↑ / HDL ↓	11.12	0.47	85.3	1.63	22
54	61735	18	21.34	regular	N	B/L PCOD	89.8	85.8	3.96	12.59	22.6	0.82	0.92	LDL ↑	7.1	0.66	216.1	1.76	21
55	61736	22	22.89	regular	N	NORMAL	121	234	4.35	21.05	27.8	2.23	1	TC ↑/LDL ↑	6.41	0.54	64.5	6.3 ↑	17
56	61737	19	21.97	long,scanty	N	NORMAL	85.2	114.8	1.96	22.28	43.4	0.78	1.08	LDL ↑ / HDL ↓	10.63	0.2	111.5	1.27	24
57	61738	19	21.34	long,scanty	A+	B/L PCOD	60	116	2.2	26.66	27.2	0.49	0.86	normal	8.93	0.6	149.3	2.57	24
58	61739	21	21.45	regular	N	NORMAL	86.5	67.9	2.61	23.37	33.1	1.35	1.16	normal	11.76	0.24	88.1	1.05	20
59	61744	19	21.87	long,scanty	A+	NORMAL	79.9	75.3	2.79	21.86	28.6	0.66	0.75	LDL ↑ / HDL ↓	7.54	2.24	116.3	0.31	25
60	61745	20	26.22	regular	A+	B/L PCOD	82.4	93.4	4.44	22.04	18.5	1.47	0.94	LDL ↑ / HDL ↓	24.08	0.08	37.08	0.94	27
61	61746	18	26.12	long,scanty	A+,AN+	B/L PCOD	68.6	89.9	15.31	201.38	4.48	1.39	0.84	normal	31.4↑	0.6	54.4	1.52	25
62	61752	19	22.77	long,scanty	N	NORMAL	80.8	73.6	3.58	26.13	22.5	0.94	0.8	normal	9.23	0.24	65.3	1.29	23
63	61753	44	22.81	regular	N	NORMAL	83.4	93.7	2.2	4.9	37.9	1.14	0.8	normal	12.73	0.5	162.8	1.77	21
64	61754	18	27.3	long,scanty	A+	B/L PCOD	84.7	105.4	8.26	59.4	10.1	2.72	0.89	normal	7.29	0.58	167.9	1.08	21
65	61755	19	22.51	long,scanty	N	NORMAL	84.8	158	1.99	6.69	42.6	1.34	1.01	LDL ↑ / HDL ↓	7.45	0.24	56.8	0.59	25
66	61757	23	22.67	long,scanty	N	NORMAL	84.7	122	2.61	21.4	32.4	1.22	1.03	normal	16.8	0.48	120.9	1	24
67	61758	22	19.17	regular	N	NORMAL	81.5	87.9	2.47	10.86	32.9	1.37	0.88	normal	19.9	0.37	73.1	2.54	25
68	61759	23	21.45	long,scanty	A+	B/L PCOD	75.9	104.2	3.65	44.98	20.5	0.95	0.7	normal	5.52	0.01	34.9	0.55	20
69	61760	20	21.56	long,scanty	N	NORMAL	91.9	84.9	2.94	69.1	31.2	0.87	0.83	LDL ↑ / HDL ↓	5.65	0.27	112.1	0.26	23
70	61761	19	21.67	long,scanty	N	NORMAL	101.2	119	1.99	10.3	50.8	0.53	0.78	LDL ↑	3.54	0.02	41.4	1.53	24
71	61762	21	21.77	regular	N	B/L PCOD	78	85.1	2.65	15.6	29.4	0.66	0.79	Normal	6.83	0.39	73.8	1.64	25

72	61763	24	21.87	long,scanty	N	NORMAL	114	81	2.36		48.3	0.85	1	Normal	6.01	0.05	55.7	0.64	23
73	61764	22	20.83	regular	N	NORMAL	76	102	3.03	47.74	25	1.21	0.88	normal	15.04	0.36	133.1	4.38	24
74	61765	21	20.96	regular	A+	B/L PCOD	75.5	97.1	3.96	12.84	19	2.85	0.87	LDL ↑ / HDL ↓	27.15	0.13	56.7	0.24	21
75	61766	20	29.3	long,scanty	A+	B/L PCOD	82	110	7	3.84	11.7	2.3	0.66	LDL ↑	13.43	0.08	41.2	2.02	23
76	61768	20	23.61	long,scanty	N	B/L PCOD	96	100	4.98	5.6	19.2	3.2	0.87	normal	21.48	0.06	37.2	2.02	25
77	61769	24	22.68	regular	N	NORMAL	85	101.4	2.36	16.04	36	1.16	0.88	normal	19.97	0.45	169.4	2.49	23
78	3644	24	18.33	long,scanty	N	NORMAL	126	124	2.81	72.99	44.8	1	0.9	TGL ↑	15.6	0.46	66.5	3.29	26
79	3645	24	19.17	long,scanty	N	NORMAL	128	80	2.23	12.86	57.3	0.94	0.71	LDL ↑	2.82	0.01	35.5	5.26↑	27
80	3647	18	19.56	regular	N	NORMAL	89	96	0.91	10.36	97.8	0.66	0.8	TGL ↑/ LDL ↑	4.98	0.1	52.5	9.34↑	16
81	3649	21	23.61	regular	N	NORMAL	91	134	2.66	1.31	34.2	0.56	0.85	LDL ↑	5.22	0.04	43.8	6.13↑	27
82	3650	19	24.56	long,scanty	A+,AN+	B/L PCOD	93	106	4.96	1.48	18.7	1.43	0.8	normal	5.99	0.09	64	6.41↑	25
83	3652	21	20.55	regular	N	NORMAL	93	104	3.52	0.85	26.4	2.48	0.83	LDL ↑	5.71	0.03	53.1	2	21
84	3653	23	22.81	long,scanty	N	NORMAL	96	115	2.04	2.65	47	0.29	0.84	LDL ↑	1.83	0.02	105.9	3.64	23
85	3655	20	22.51	long,scanty	A+	B/L PCOD	93	71	5.93	12.14	15.6	2.99	1.08	LDL ↑	5.91	0.04	76.6	1.92	24
86	3656	18	27.39	regular	A+	B/L PCOD	95	89	8.33	38.24	11.4	1.02	0.76	TGL ↑/ LDL ↑	4.82	0.04	56	1.92	25
87	3658	22	21.77	long,scanty	N	NORMAL	92	119	3.9	13.66	23.5	2.11	1.06	normal	32.7	0.31	103.6	2.47	18
88	3660	18	20.4	long,scanty	N	NORMAL	97	82	2.24	15.16	43.3	1.36	0.85	LDL ↑	14.4	0.04	39.5	1.71	16
89	3661	24	26.63	long,scanty	A+	B/L PCOD	100	136	9.64	25.7	10.3	0.64	1.06	normal	7.67	0.09	98.7	2.01	24
90	3663	22	19.37	long,scanty	N	NORMAL	94	82	4.23	88.7	23.6	0.93	0.84	normal	8.04	0.3	40.2	3.13	27
91	3666	24	27.19	regular	A+,AN,MILD H+	B/L PCOD	87	78	9.37	3.21	9.2	0.96	1.18	LDL ↑	8.76	0.12	49.7	3.51	14
92	3646	18	21.22	long,scanty	N	B/L PCOD	99	102	2.7	10.22	13.5	1.17	0.88	LDL ↑	7.6	0.11	42.6	11.14 ↑	28
93	3648	20	22.68	long,scanty	A+	B/L PCOD	94	76	9.56	164.6	9.8	1.33	0.82	TGL ↑/ LDL ↑	4.02	0.04	39.4	1.38	18
94	3651	20	18.33	long,scanty	N	NORMAL	95	100	3.89	5.68	24.4	0.89	1.1	normal	5.67	0.12	55.7	3.02	15
95	3654	18	21.77	regular	N	B/L PCOD	102	80	5.38	33.3	18.9	0.73	0.86	normal	4.85	0.03	84.2	4.23	24
96	3657	18	20.96	long,scanty	A+	B/L PCOD	83	104	6.29	14.02	13.1	1.66	0.87	normal	5.18	0.12	44.4	6.69	25
97	3659	21	18.33	long,scanty	N	NORMAL	88	71	2.98	4.6	29.5	1.45	1.09	Normal	3.3	0.07	84	4.2	24
98	3662	20	21.09	regular	N	NORMAL	107	118	2.93	12.5	36.5	1.06	1.04	normal	4.6	0.07	74.5	9.52	28
99	3664	19	20.96	long,scanty	N	B/L PCOD	86	107	4.12	29.3	20.8	3.28	0.88	normal	9.53	0.35	50.7	35.14 ↑	21
100	3667	19	19.91	regular	N	NORMAL	87	72	2.89	4.37	30.1	1.33	0.97	normal	7.23	0.12	55.7	2.91	26
101	3668	20	20.08	regular	N	NORMAL	95	131	2.02	15.06	47	2.33	1.05	normal	18.57	0.17	46.2	5.33 ↑	18
102	12603	19	24.14	regular	A+,AN+,MILD H+	B/L PCOD	91.2	71.3	11.12	39.69	8.2	0.92	1.03	normal	9.38	0.19	152.9	3.74	25
103	12604	22	18.55	regular	A+	B/L PCOD	92.2	72.3	5.51	10.11	16.7	1.47	0.88	normal	13.88	0.49	168.9	3.33	26
104	12606	20	21.09	regular	A+	B/L PCOD	90.8	81.6	9.22	7.03	9.8	1.02	0.88	normal	10.55	0.39	36.3	6.71↑	17
105	12608	18	20.96	long,scanty	N	NORMAL	100	86.3	3.11		32.1	0.82	0.87	normal	6.79	0.36	132.2	7.95↑	25
106	12611	21	21.56	long,scanty	A+	B/L PCOD	85.6	104.7	7.28	95.23	11.7	0.88	0.92	normal	13.6	0.53	156.6	7.2	23
107	12612	19	18.97	long,scanty	N	B/L PCOD	79.6	95.9	5.15	24.35	15.4	3.02	1.08	normal	7.37	0.33	74	2.2	25
108	12613	22	19.17	regular	N	NORMAL	87.1	80.09	3.81	17.18	22.8	0.91	0.9	normal	24.5	0.44	125.5	0.8	13

109	12614	20	18.55	long,scanty	N	B/L PCOD	95.8	84.3	4.74	37.85	20.2	1.75	0.84	normal	8.54	0.18	82.9	1.06	27
110	12616	20	18.77	regular	N	NORMAL	85.1	90.3	2.18	27.83	39	0.87	0.94	normal	16.17	0.25	101.9	1.14	29
111	12617	19	19.56	long,scanty	N	B/L PCOD	95.6	96.07	3.51	15.77	27.2	1.47	1.06	normal	8.33	0.23	156.2	1.14	21
112	12618	22	20.55	regular	N	NORMAL	90.8	98.7	2.73	12.92	33.2	1.24	0.88	normal	6.83	0.4	174.8	0.77	25
113	12619	22	21.22	regular	N	NORMAL	91.1	131.9	2.73	12.92	33.3	1.24	0.88	LDL ↑	10.32	0.29	79.9	0.49	22
114	12620	19	21.34	regular	A+	B/L PCOD	89.09	82.4	4.77	7.02	18.6	1.95	0.97	normal	5.53	0.26	95.4	1.78	23
115	12621	19	26.12	long,scanty	A+,AN+	B/L PCOD	84.5	109.4	11.7	29.46	7.2	1.36	0.85	normal	14.4	0.04	39.5	1.71	24
116	12622	20	19.17	regular	N	NORMAL	84	77.4	4.31	2.32	19.4	1.79	1.15	normal	17.8	0.41	104.2	2.6	18
117	12623	20	20.69	long,scanty	A+	B/L PCOD	95.2	119.2	6.9	79.2	13.7	2.98	0.97	normal	8.99	0.53	153.9	2.3	24
118	12624	20	21.09	regular	A+,AN+	B/L PCOD	90.3	167	10.69	76.24	8.4	1.68	1.02	LDL ↑	7.98	0.43	200.4	0.29	25
119	12625	24	21.34	regular	A+	B/L PCOD	88.1	120	8.53	6.03	10.3	0.96	1.12	normal	5.09	0.26	86.2	2	15
120	12626	18	20.69	long,scanty	N	NORMAL	95.8	84.3	2.18	27.3	43.9	0.87	0.94	normal	16.1	0.26	101.9	1.14	22
121	12627	21	20.4	long,scanty	N	NORMAL	99.3	120	3.5	16.06	28.3	1.52	0.82	normal	26.56	0.62	290	0.64	24
122	12628	21	21.67	long,scanty	N	NORMAL	81.6	88.8	2.45	12.49	33.3	4.3	0.8	normal	10.01	0.16	74.3	0.7	26
123	12629	20	20.96	regular	N	NORMAL	83.1	91.5	2.41	3.07	34.4	0.74	0.9	LDL ↑	16.09	0.24	107.8	0.8	22
124	12630	22	20.83	regular	N	B/L PCOD	82.1	129.7	5.8	26.7	14.1	1.14	1.03	normal	6.31	0.21	169.8	0.43	26
125	12631	20	21.08	regular	N	B/L PCOD	90.1	109.1	6.91	36.99	13	1.19	0.94	normal	16.94	0.15	73.2	2.4	15
126	12632	19	22.37	long,scanty	N	B/L PCOD	85.2	99.4	5.71	25.98	14.9	0.5	1.11	LDL ↑	12.57	0.39	204.2	0.9	23
127	12633	21	18.97	long,scanty	N	NORMAL	90.1	51.8	4.12	17.67	21.8	1.76	0.94	normal	15.07	0.61	97.7	1.83	25
128	12635	21	19.91	menorrhagia	N	B/L PCOD	85.2	81.5	4.14	7.92	20.5	0.64	0.89	normal	20.29	0.3	144.5	1.13	22
129	12636	20	18.55	long,scanty	A+	B/L PCOD	88.1	145	6.97	86.27	12.6	2.15	0.81	normal	7.26	0.19	131.9	0.68	25
130	12637	21	21.22	regular	A+,AN+	B/L PCOD	89.8	99.5	4.76	27.27	18.8	0.85	1.28	normal	7.76	0.46	152.6	1.63	14
131	12638	18	20.55	long,scanty	A+	NORMAL	99.4	177.4	15.46	31.5	6.429	1.51	0.9	LDL ↑ / HDL ↓	6.83	0.11	79	1.28	22
132	12639	20	22.94	long,scanty	A+	B/L PCOD	88.9	107.3	6.38	42.5	13.9	0.5	1.1	normal	9.32	0.35	186.4	1.38	13
133	12640	22	25.2	long,scanty	A+,AN+	B/L PCOD	87.4	82.3	9.35	37.33	9.3	1.43	0.88	normal	8.29	0.32	170	0.5	28
134	12641	18	26.3	regular	A+	B/L PCOD	98.7	119.1	9.4	37.33	10.5	1.43	0.88	LDL ↑ / HDL ↓	8.29	0.32	121.2	0.53	25
135	35269	23	20.4	long,scanty	N	B/L PCOD	102	112	3.8	9.01	26.8	1.95	0.85	normal	13.31	0.28	142.3	0.3	22
136	35270	18	18.97	regular	N	NORMAL	75	83	2.1	6.5	35.7	1.2	0.77	normal	4.83	0.19	74.5	0.74	26
137	35271	18	20.96	long,scanty	N	B/L PCOD	71	83	4.8	13.65	14.7			normal					28
138	35272	21	21.67	regular	N	NORMAL	79	92	2.23	6.45	35.4	0.96	0.77	L ↑ /LDL↑/ HI	4.47	0.13	70.3	0.36	17
139	35273	18	19.56	long,scanty	N	NORMAL	99	108	1.95	3.05	50.7	0.74	0.62	normal	5.76	0.02	40	0.39	12
140	35274	18	21.87	regular	N	NORMAL	89	103	1.9	5.6	46.8	0.87	0.94	normal	4.62	0.02	50.1	0.68	16
141	35275	18	21.09	long,scanty	N	NORMAL	76	102	1.47	5.7	51.7	0.38	2.56	normal	3.02	0.02	35.6	1.54	24
142	35276	19	21.22	long,scanty	N	B/L PCOD	83	101	7.69	22.6	10.7	0.67	0.9	normal	5.4	0.1	62.7	0.47	16
143	35277	20	22.37	regular	N	NORMAL	101	123	2.1	6.7	48	0.61	0.99	normal	4.69	0.11	59.6	0.62	29
144	35278	23	26.38	long,scanty	A+	B/L PCOD	78	89	12.08	38.9	6.45	0.93	0.99	LDL ↑ / HDL ↓	4.93	0.14	41	0.47	23
145	35279	18	24.24	regular	A+,AN+	B/L PCOD	73	102	6.33	7.5	11.5	0.56	0.77	normal	1.57	0.04	37	0.37	32

146	35280	18	19.56	long,scanty	N	NORMAL	78	99	1.95	2.65	40	1.39	0.95	normal	1.52	0.12	56.9	0.45	19
147	35281	23	21.56	long,scanty	N	B/L PCOD	74	102	5.36	10.98	4.55	0.93	0.9	normal	1.36	0.1	39	0.26	28
148	35282	24	20.69	regular	A+,AN+	B/L PCOD	87	87	16.24	7.83	16.2	1.1	0.9	↓↑/LDL↑/ HI	2.44	0.16	41.9	0.91	30
149	35283	22	22.15	long,scanty	N	B/L PCOD	89	110	3.94	12.4	22.5	1.52	0.85	normal	11.8	0.36	163.2	0.86	24
150	35284	18	20.24	regular	N	NORMAL	89	93	1.91	3.33	46.5	0.71	0.88	normal	2.73	0.18	37	0.63	26
151	35285	24	26.1	regular	N	B/L PCOD	89	111	8.17	24.7	10.8	0.74	0.92	normal	3.45	0.01	57.3	0.65	22
152	35286	18	19.37	regular	N	NORMAL	79	123	1.97	3.88	40.1	0.78	0.88	normal	12.59	0.03	39.9	0.76	28
153	35287	19	21.34	long,scanty	N	B/L PCOD	76	113	7.36	8.62	10.3	1.76	0.92	TGL ↑ / HDL ↓	3.91	0.14	35.3	0.7	11
154	35288	23	20.69	long,scanty	N	B/L PCOD	76	110	18.9	5.9	4.02	1.17	1.03	LDL ↑	11.22	0.26	41.9	0.61	21
155	35289	24	19.74	regular	N	NORMAL	85	103	2.56	7.94	33.2	0.42	0.76	normal	1.56	0.08	39.1	0.41	27
156	35290	21	20.83	long,scanty	A+	B/L PCOD	76	89	4.22	9.74	18	1.04	1.2	LDL ↑	7.68	0.67	362.6	0.58	23
157	35291	19	22.94	regular	N	NORMAL	92	110	3.63	19.76	25.3	1.49	1	normal	15.15	0.04	36.8	1.19	23
158	35292	21	21.4	long,scanty	N	NORMAL	79	102	2	8.46	39.5	1.14	1.03	normal	6.31	0.21	169.8	0.43	26
159	35293	22	22.68	long,scanty	N	B/L PCOD	79	109	5.82	24.46	13.5	1.19	0.77	normal	17.29	0.36	155.4	0.25	15
160	35294	19	24.74	regular	A+,AN+,MILD H+	B/L PCOD	99	122	10.9	20.01	9	1.45	0.81	TGL ↑	36.02↑	0.5	194.7	0.33	26
161	35295	18	20.55	long,scanty	A+	NORMAL	75	101	1.97	18.98	38	1.07	0.82	normal	3.71	0.38	85.7	0.76	22
162	35296	20	19.37	regular	N	NORMAL	86	119	4.11	32.11	20.9	1.02	0.89	normal	4.49	0.67	167.5	0.68	25
163	35297	21	21.65	long,scanty	A+	B/L PCOD	88	119	8.05	34.3	10.9	0.51	0.78	normal	10.43	0.25	124.2	0.79	15
164	35298	23	21.43	regular	A+	B/L PCOD	77	83	6.36	19.9	12.1	2.3	0.87	normal	15.05	0.32	155.7	0.81	24
165	35299	18	19.47	long,scanty	A+,AN+	B/L PCOD	95	115	8.9	32.9	10.6	4	0.8	LDL ↑ / HDL ↓	11.09	0.64	136.6	0.84	25
166	35561	22	18.76	regular	N	NORMAL	91	115	1.99	5.59	45.7	0.96	0.88	normal	11.09	0.4	166.4	1.19	15
167	35564	23	19.56	long,scanty	A+	B/L PCOD	93	104	7.38	21.52	12.6	1.95	0.88	normal	13.76	0.18	102.6	0.83	23
168	35566	20	19.37	long,scanty	A+	B/L PCOD	91	106	5.19	17.47	17.5	1.78	0.88	normal	28.95	0.52	303.7	2.14	21
169	35569	21	20.69	long,scanty	N	NORMAL	104	126	2.22	3.6	46.8	1.18	0.89	LDL ↑	10.5	0.48	105.8	2.5	20
170	35573	18	25.21	regular	N	B/L PCOD	95	115	16.03	23.71	5.93	22.01 ↑	0.61	LDL ↑	29.74	0.37	82.3	0.81	27
171	35575	23	22.15	regular	N	NORMAL	86.4	93	3.2		27	2.09	0.85	LDL ↑ / HDL ↓	14.05	0.48	158.1	1.89	21
172	35578	21	21.56	long,scanty	N	B/L PCOD	85.2	114.8	7.24		11.17	2.21	0.99	normal	13.82	0.59	153.3	0.92	22
173	35581	22	20.56	regular	N	NORMAL	79.9	75.3	4.13	4.28	19.3	2.76	0.86	normal	21.48	0.37	218.7	0.4	15
174	35583	18	23.28	long,scanty	A+	B/L PCOD	77	100	9.83	3.01	7.8	1.27	1.02	normal	18.94	0.46	299.4	4.5	27
175	35586	21	19.74	long,scanty	N	NORMAL	102	140	5.62	10.6	18.1	3.4	0.97	TGL ↑	17.99	0.42	83.6	1.93	26
176	35588	21	22.15	regular	A+	B/L PCOD	109	110	8.77	27.32	12.4	1.74	0.69	TGL ↑	25.9	0.39	97.3	0.92	16
177	35589	19	20.61	long,scanty	N	B/L PCOD	89.4	99.6	5.79	6.3	15.4	2.18	1.24	LDL ↑	15.2	0.04	389.2	0.44	16
178	35592	21	21.9	long,scanty	A+	B/L PCOD	102	111	2.23	6.96	45.7	1.93	0.9	normal	20.53	0.49	199.8	0.5	22
179	35594	18	24.76	regular	A+	B/L PCOD	80	103	10.21	41.92	7.83	2.57	0.86	normal	4.78	0.38	82.1	0.59	23
180	35598	18	22.67	long,scanty	N	NORMAL	86	108	4.98	42.6	17.2	1.76	0.72	normal	14.02	0.15	80.1	0.74	21
181	35604	21	22.51	regular	N	NORMAL	76	94	7.19	7.41	10.5	1.65	0.94	normal	22.82	0.46	182.3	1.28	26
182	35607	22	18.65	long,scanty	N	NORMAL	89	95	4.6	9.26	19.3	1.79	0.91	normal	9.6	0.46	145	0.65	26

183	35616	18	19.74	long,scanty	A+,AN+	B/L PCOD	94	110	5.75	17.94	16.3	2.9	0.98	normal	20.56	0.03	112	1.73	24
184	35619	22	21.5	long,scanty	N	NORMAL	78	113	3.46	15.8	22.5	1.06	0.85	normal	10.02	0.5	71.8	1.54	15
185	35623	24	21.08	regular	N	B/L PCOD	89	114	5.12	8.69	17.3	1.79	1.03	normal	8.32	0.02	202.1	0.96	24
186	35627	19	21.45	long,scanty	N	NORMAL	67	116	3.81	8.23	17.5	1.67	0.84	normal	39.5 ↑	0.3	56	0.36	21
187	35629	23	23.28	long,scanty	A+	B/L PCOD	95	118	21.32	6.36	4.45	1.89	0.83	LDL ↑	10.73	0.09	46.8	0.36	24
188	35634	18	26.35	long,scanty	A+	B/L PCOD	75	108	11.88	21.27	6.3	0.77	0.93	normal	9.76	0.04	406.3	0.27	14
189	35637	22	21.43	regular	A+,AN+,MILD H+	B/L PCOD	114	162	8.24	32.43	13.8	1.43	0.94	LDL ↑ / HDL ↓	21.8	0.03	151.1	0.14	25
190	35639	20	22.15	long,scanty	N	NORMAL	86	100	2.07	1.56	41.5	0.65	0.85	normal	8.96	0.07	62.5	0.7	15
191	35643	19	19.52	regular	N	NORMAL	116	148	5.07	4.75	22.8	1.88	0.97	LDL ↑	19	0.17	175.8	0.95	22
192	35646	19	22.12	long,scanty	N	NORMAL													26
193	35649	24	22.15	regular	N	B/L PCOD	92	113	2.28	1.71	40.3	1.81	0.82	normal	8.12	0.28	84.6	0.36	25
194	35653	19	21.56	long,scanty	A+	B/L PCOD	76	106	4.8	16.98	15.8	2.09	0.75	normal	7.75	4.48	121.7	0.82	22
195	35654	23	20.4	long,scanty	N	NORMAL	96.1	102.4	2.58	10.11	37.2	0.66	0.99	normal	7.8	0.32	46.2	0.52	23
196	35662	22	21.34	long,scanty	N	NORMAL	94	128	3.61	2.92	26	2.73	1.03	normal	11.39	0.35	73.1	0.86	16
197	35668	19	24.37	regular	N	B/L PCOD	75	102	18.12	42.6	4.13	2.65	0.87	Normal	5.23	0.37	76.3	1.16	23
198	41927	22	21.56	long,scanty	N	NORMAL	84	106	3.96	4.47	21.4	3.24	0.58	Normal	11.3	0.35	51.1	1.11	16
199	41928	23	19.91	long,scanty	N	B/L PCOD	88.6	90	3.44	15.5	25.7	2.34	0.82	Normal	12.41	0.11	68.8	0.66	20
200	41929	18	21.22	regular	N	NORMAL	86	120	2.56	23.17	33.5	0.56	1.01	Normal	13.14	0.07	50.6	0.53	20
201	41930	24	20.55	regular	N	NORMAL	85.2	114	2.78	26.7	30.6	1	0.97	Normal	11.85	0.53	60.7	0.26	23
202	41931	20	21.67	long,scanty	N	NORMAL	82	110	2.58	10.41	31.7	0.66	0.77	Normal	7.88	0.3	49.6	0.5	23
203	41932	20	20.96	regular	N	NORMAL	76	94	2.87	2.6	26.4	0.6	0.91	Normal	7.22	0.19	42.6	0.41	20
204	41933	21	19.56	long,scanty	N	B/L PCOD	95	100	3.66	6.76	25.9	1.45	0.88	Normal	16.69	0.29	44.6	0.84	23
205	41934	23	22.91	long,scanty	A+	B/L PCOD	87	118	9.91	13.6	8.7	0.99	0.9	LDL ↑ / HDL ↓	16.48	0.25	74	0.72	26
206	41935	20	21.9	long,scanty	N	NORMAL	95.2	119	3.73	12.83	25.5	0.83	0.9	Normal	17.15	0.36	101.7	0.38	16
207	41936	18	26.11	regular	A+	B/L PCOD	71.3	115	9.12	33.91	7.8	1.6	0.65	LDL ↑	16.51	0.19	66.4	0.28	16
208	41937	22	25.21	long,scanty	N	B/L PCOD	81	100	18.1	62.53	4.48	2.29	0.88	TGL ↑ / LDL ↑	23.06	0.21	37.9	0.19	23
209	41938	24	20.83	regular	N	NORMAL	94.4	110	2.29	8.79	41.2	0.9	0.68	Normal	10.44		66.9	0.81	22
210	41939	23	20.55	long,scanty	N	NORMAL	92.9	109.4	3.48	6.02	26.6	0.66	0.99	Normal	17.06	0.71	179.2	0.55	24
211	41945	20	21.75	long,scanty	N	NORMAL	113	115	1.91	5.7	59.1	1.45	1.04	Normal	18.4	0.42	72.2	1.07	26
212	41946	19	19.47	long,scanty	N	NORMAL	84.7	78.8	1.94	4.03	43.6	2.71	0.82	Normal	9.81	0.2	36.1	1.1	20
213	41947	21	20.42	regular	N	B/L PCOD	84.7	105.4	9.55	30.64	8.86	5.79 ↑	0.97	Normal	5.31	0.16	52.6	0.63	25
214	41948	21	20.96	long,scanty	A+	B/L PCOD	79.5	100.7	4.23	13.68	18.7	7.54 ↑	0.87	Normal	25.6	0.35	169.8	0.67	19
215	41949	20	24.43	long,scanty	A+	B/L PCOD	84.4	110.7	9.77	19.54	8.6	0.44	1.1	Normal	7.93	0.23	55.8	0.81	24
216	41950	21	19.85	regular	N	NORMAL	84.8	73.6	1.93	4.32	43.9	1.53	0.97	Normal	4.85	0.29	54.3	0.71	23
217	41951	23	23.86	regular	N	B/L PCOD	82.9	109	9.63	31.78	8.6	0.46	0.9	Normal	2.34	0.33	186.8	1.92	18
218	41952	19	21.07	regular	N	NORMAL	88	112	1.58	70.3	55.6	1.19	0.82	Normal	3.47	0.38	162.8	0.76	24
219	41953	20	18.75	long,scanty	N	NORMAL	81.5	87.9	2.24	6.53	36.3	12.04↑	0.48	LDL ↑	5.78	0.25	101.4	0.62	24

220	41954	18	19.17	regular	A+	B/L PCOD	79.5	100.7	5.7	20.8	13.9	0.84	1.01	Normal	11.48	0.04	56.6	0.19	26
221	41955	19	19.9127.32	long,scanty	A+	B/L PCOD	84.7	94.5	21.5	108.08	3.9	0.84	1.01	LDL ↑	11.48	0.04	56.6	0.81	16
222	41956	21	22.94	long,scanty	N	NORMAL	97.8	121.6	2.19	14.21	44.6	1.51	0.84	Normal	9.11	0.34	50.6	0.6	23
223	35656	20	19.17	regular	N	NORMAL	102	112	3.8	7.89	26.8	1.05	1.08	normal	27.78	0.29	71	1.09	24
							F (mg/dl)	PP (mg/dl)	F (uIU/ml)	PP (uIU/ml)									